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THE TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY

OF EDINBURGH

VOL. XXXIII.—NEW SERIES

SESSION 1913-1914

1914

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JAMES THIN, PUBLISHER TO THE SOCIETY
Edinburgh

PREFACE

THE present Volume is the *Thirty-third* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

Ten meetings were held in the year, two being clinical meetings.

CHALMERS WATSON,
Editor.

October 1914.

Medico-Chirurgical Society of Edinburgh.

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	Alfred Nolan Bell, M.D., M.R.C.P.L., *Colchester*, . .	1907
	William Mackirdy, L.R.C.P.L., L.R.C.S.L., *Peterborough*,	1907
470	James Bartholomew Mears, L.R.C.P. & S.L., *Lanchester*, .	1907
	F. Pattison, M.B., L.R.C.P., *Kirkcaldy*, . . .	1907
	J. H. Douglas Webster, M.B., Ch.B., . . .	1907
	Louis Lourie, M.B., Ch.B., *South Africa*, . . .	1908
	Alex. Dun, M.B., F.R.C.S., *Dundee*, . . .	1909
475	Rae Gibson, M.B., M.R.C.P.L., ...	1910
	J. C. Thomson, M.D., *Dumfries*, ...	1910
	James Lochhead, M.D., F.R.C.S., *Gibraltar*, . .	1911
	Major A. N. Fleming, R.A.M.C., *London*, . .	1912
	D. Haig, M.B., C.M., *Crieff*, ...	1912

ORDINARY MEMBERS.

ARRANGED ALPHABETICALLY.

(*a*) Members who pay the Annual Subscription.

		Date of Admission.
	Dr R. Abernethy, 10 St Colme Street,	1892
	Sir J. O. Affleck, 38 Heriot Row,	1871
	Dr A. C. Ainslie, 49 Minto Street,	1898
	Dr R. S. Aitchison, Woodhurstleigh, Crawley, Sussex,	1887
5	Dr R. Charles Alexander, 15 Hartington Gardens,	1913
	Dr J. L. Annan, 27 Braid Road,	1913
	Dr E. F. Armour, 6 Bruntsfield Terrace,	1890
	Dr Harold S. Ballantyne, Dalkeith,	1899
	Dr J. W. Ballantyne, 19 Rothesay Terrace,	1885
10	Dr A. H. Freeland Barbour, 4 Charlotte Square,	1881
	Lewis Beesly, Esq., 42 Northumberland Street,	1905
	Dr G. A. Berry, 31 Drumsheugh Gardens,	1883
	Dr Robert H. Blaikie, 5 Mayfield Gardens,	1883
	William Booth, Esq., 2 Minto Street,	1888
15	Dr Philip G. Borrowman, 74 Great King Street,	1893
	Dr John M. Bowie, 53 Grange Road.	1901
	Dr F. D. Boyd, C.M.G., 22 Manor Place,	1891
	Dr Byrom Bramwell, 23 Drumsheugh Gardens,	1876
	Dr Edwin Bramwell, 24 Walker Street, *Secretary*,	1900
20	Dr N. T. Brewis, 6 Drumsheugh Gardens,	1886
	Dr A. J. Brock, 27 Braid Crescent,	1907
	Dr J. J. Graham Brown, 3 Chester Street,	1878
	Dr R. Dods Brown, Murray House, Perth,	1908
	Dr A. Ninian Bruce, 8 Ainslie Place,	1913
25	Dr W. H. Bryce, Lathallan House, Colinsburgh,	1911
	Dr A. H. Buchan, 2 Dean Terrace,	1899
	Dr Buist, 1 Clifton Terrace,	1877
	Dr T. M. Burn-Murdoch, 14 Charlotte Square,	1886
	Dr J. Burnet, 6 Glengyle Terrace,	1907
30	Professor Francis M. Caird, 13 Charlotte Square,	1883
	Dr H. L. Calder, 60 Leith Walk, Leith,	1884
	Dr James Cameron, 71 Newington Road,	1895
	W. W. Carlow, Esq., 20 Eglinton Crescent,	1912
	Dr A. N. S. Carmichael, 2 Merchiston Avenue,	1902
35	Dr Edward Carmichael, 21 Abercromby Place,	1887
	Dr E. W. Scott Carmichael, 40 Palmerston Place,	1901
	Dr George Scott Carmichael, 25 Braid Road,	1903
	Dr N. Scott Carmichael, 44 Polwarth Terrace,	1910
	Dr George J. R. Carruthers, 4A Melville Street,	1901
40	C. W. Cathcart, Esq., 44 Melville Street,	1883
	Dr J. G. Cattanach, 3 Alvanley Terrace,	1895
	George L. Chiene, Esq., 23 Alva Street,	1901
	Emeritus Professor John Chiene, C.B., 21 Alva Street,	1867
	A. E. Chisholm, Esq., 7 Claremont Crescent,	1910
45	Dr H. M. Church, 28 Cluny Drive,	1876
	Dr A. W. Greenhorn Clark, 25 Greenbank Crescent,	1908
	Dr R. D. Clarkson, Larbert Institution, Larbert,	1896
	Sir T. S. Clouston, 26 Heriot Row,	1861
	Dr John D. Comrie, 25 Manor Place,	1902
50	Dr W. E. Cooke, Ochil Hills Sanatorium,	1914
	Dennis Cotterill, Esq., 10 Coates Crescent,	1909
	J. M. Cotterill, Esq., 24 Manor Place,	1878
	Dr John Craig, 216 Bruntsfield Place,	1900
	Dr William Craig, 71 Bruntsfield Place,	1869

		Date of Admission.
55	Dr D. M. Crichton, Burgh House, Maryhill, Glasgow,	1906
	Dr David Halliday Crom, 17 Alva Street,	1906
	Professor Sir John Halliday Croom, 25 Charlotte Square,	1870
	Dr W. J. Crow, 108 Mannofield Road,	1908
	Dr John Cumming, 70 Braidshell Place,	1894
60	Dr T. B. Darling, 13 Merchiston Place,	1887
	Dr Michael Dewar, 24 Lauriston Place,	1885
	Dr Thomas W. Dewar, Kinnaird, Dunblane,	1906
	Dr J. K. Milne Dickie, 37 Lauriston Place,	1912
	Dr J. T. Dickie, 37 Lauriston Place,	1900
65	Dr D. Flint Dickson, Hill Crescent, Lochgelly, Fife,	1906
	Dr George Dickson, 26 Merchiston Park,	1901
	Dr W. E. Carnegie Dickson, 100 Findhorn Place,	1902
	Dr C. E. Douglas, Cupar-Fife,	1894
	Dr Kenneth M. Douglas, 19 Chester Street,	1888
70	J. W. Dowden, Esq., 48 Manor Place,	1893
	Dr W. B. Drummond, 2 Brandon Terrace,	1900
	Dr John A. H. Duncan, 4 Cluny Drive,	1895
	Dr H. M. Dunlop, 20 Abercromby Place,	1883
	Dr J. C. Dunlop, 33 Chester Street,	1892
75	Dr John Eason, 58 Northumberland Street,	1900
	Dr William Elder, 4 John's Place, Leith,	1892
	Dr J. Haig Ferguson, 7 Coates Crescent,	1885
	Dr Thomas Finlay, 8 Comely Bank Road,	1903
	Dr Andrew F. Fleming, St John's Road, Corstorphine,	1905
80	Dr R. A. Fleming, 10 Chester Street,	1890
	Dr A. B. Flett, 15 Walker Street	1903
	Dr W. L. Foggie, 4 Airlie Place, Dundee,	1907
	Dr A. Dingwall Fordyce, 8 Manor Place,	1901
	Dr W. Fordyce, 20 Charlotte Square,	1907
85	Dr Hope Fowler, 21 Walker Street,	1913
	Dr J. S. Fowler, 55 Northumberland Street,	1895
	Dr John Fraser, 54 Great King Street,	1878
	John Fraser, Esq., 3 Darnaway Street,	1911
	Dr J. Hossack Fraser, Fernbell, Bridge of Allan,	1895
90	Dr J. S. Fraser, 50 Melville Street,	1905
	Prof. Sir Thomas R. Fraser, 13 Drumsheugh Gardens,	1865
	Dr W. Ernest Frost, 36 Moray Place,	1904
	Dr G. Fyfe, 24 Broughton Place,	1911
	Dr F. Gardiner, 9 George Square,	1900
95	Dr J. H. Gibbs, 12 Coates Crescent,	1905
	Dr A. B. Giles, 4 Palmerston Place,	1893
	Dr Alexander Goodall, 14 Walker Street,	1901
	Dr D. J. Graham, 2 Melville Crescent,	1897
	Dr F. M. Graham, 16 Mayfield Gardens,	1894
100	Dr J. M. Graham, 6 Castle Terrace,	1908
	Dr J. Taylor Grant, 3 Manor Place,	1898
	Dr J. Allan Gray, 107 Terry Road,	1879
	Dr John Alexander Gray, 4 Walseley Terrace,	1911
	Dr John L. Green, 23 Minto Street,	1904
105	Emeritus Professor Greenfield, Kirkbrae, Elie, Fife,	1886
	Dr David M. Greig, 25 Tay Street, Dundee,	1892
	Dr G. L. Gulland, 11 Chester Street,	1888
	Dr A. C. Guthrie, 21 Piling Street,	1890
	Dr John Guy, Public Health Department, Edinburgh,	1913
110	Dr William Guy, 11 Wemyss Place,	1890
	Dr William Haldane, Viewforth, Bridge of Allan,	1880
	Dr D. Berry Hart, 5 Randolph Cliff,	1886
	Dr James Harvey, 7 Blenheim Place,	1893
	Dr A. Fergus Hewat, 3 Darnaway Street,	1911
115	J. W. B. Hoololon, Esq., 6 Chester Street, *Vice-President*,	1883
	Dr George Hunter, 33 Palmerston Place,	1876

		Date of Admission.
	Dr R. W. Inkster, 61 Windsor Street,	1895
	Dr Alex. James, 14 Randolph Crescent,	1877
	Dr Allan Jamieson, 35 Charlotte Square,	1876
120	Dr Hugh Jamieson, 1 Strathearn Road,	1889
	Dr J. Boyd Jamieson, 43 George Square,	1900
	Dr Frank E. Jardine, 83 Great King Street,	1911
	Dr G. M. Johnston, 7 Wellington Place, Leith,	1888
	Dr R. J. Johnston, 18 George Square,	1897
125	Dr R. W. Johnstone, 10 Alva Street,	1907
	Dr C. B. Ker, City Hospital, Comiston Road,	1894
	Dr F. K. Kerr, 168 Newhaven Road, Leith,	1910
	Dr George Kerr, 6 St Colme Street,	1885
	Dr J. Lamond Lackie, 7 Randolph Crescent,	1907
130	Dr H. G. Langwill, 4 Hermitage Place, Leith,	1899
	Dr Jas. Langwill, Kirkcaldy,	1912
	Dr David Lawson, Nordrach-on-Dee, Banchory,	1901
	Dr H. A. Leehody, Corstorphine,	1899
	Dr Edwin M. Lithgow, 8 Alva Street,	1901
135	Dr John D. Lithgow, 8 Alva Street,	1901
	Professor Harvey Littlejohn, 11 Rutland Street,	1889
	Dr R. Cranston Low, 7 Castle Terrace,	1906
	Dr T. D. Luke, Hydropathic, Peebles,	1913
	Dr R. A. Lundie, 55A Grange Road,	1883
140	Dr W. Leslie Lyall, 8 Murrayfield Gardens,	1902
	Dr D. C. A. M'Allum, 11 Greenhill Gardens,	1900
	Dr Angus Macdonald, 42 Polwarth Terrace,	1902
	Dr John M'Gibbon, 24 Melville Street,	1899
	Dr C. W. MacGillivray, 15 Charlotte Square,	1877
145	Dr A. M. M'Intosh, 30 East Claremont Street,	1904
	Dr Archibald Mackintosh, 50 Trinity Road,	1911
	Dr George Mackay, 20 Drumsheugh Gardens,	1887
	Dr G. Reginald Mackay, 5 Walker Street,	1913
	Dr A. M'Kendrick, 27 Chalmers Street,	1909
150	Dr W. Leslie Mackenzie, 1 Stirling Road, Trinity,	1895
	Dr Malcolm M'Larty, 22 Abercromby Place,	1900
	Dr C. M'Neil, 23 Stafford Street,	1909
	Dr John Macpherson, 8 Darnaway Street,	1891
	Dr John MacRae, Lynwood, Murrayfield Road,	1893
155	Dr Douglas M'Rae, Glengall House, Ayr,	1904
	Dr Alexander M. Malcolmson, St John's Road, Corstorphine,	1905
	Dr A. A. Matheson, 41 George Square,	1901
	Dr Roderick M. Matheson, 33 Buccleuch Place,	1902
	Dr George D. Mathewson, 5 Castle Terrace,	1911
160	Dr Edwin Matthew, 9 Walker Street,	1900
	Dr J. Halley Meikle, 12 Midmar Gardens,	1906
	Dr Kenmure Melville, 2 Nile Grove,	1908
	A. Miles, Esq., 20 Walker Street,	1892
	Dr J. Mill, 188 Ferry Road,	1886
165	Dr Wilkie Millar, 6 Blenheim Place,	1913
	A. G. Miller, Esq., 8 Garscube Terrace,	1867
	Dr James Miller, 5 Hermitage Terrace	1910
	Dr W. H. Miller, 51 Northumberland Street,	1895
	Dr W. Morrison Milne, 3 Minto Street,	1905
170	Dr A. P. Mitchell, 25 Montrose Terrace,	1912
	Dr R. S. Mowat, 40 Raeburn Place,	1912
	Dr T. Goodall Nasmyth, 27 Palmerston Place,	1884
	Dr H. Oliphant Nicholson, 20 Manor Place,	1900
	Dr John Orr, 6 Strathearn Road,	1895
175	Dr W. Basil Orr, 13 Braid Road,	1893
	Dr G. Keppie Paterson, 19 Albany Street,	1889
	Dr J. V. Paterson, 11 Melville Street,	1900
	Dr Simla Paterson, 147 Bruntsfield Place,	1904

		Date of Admission
	Dr William Patterson, 8 Hermitage Place, Leith,	1909
180	Dr C. M. Pearson, 14 Manor Place,	1902
	Dr John H. H. Pearson, 12 Primrose Bank Road, Trinity,	1911
	Dr H. A. Peddie, 24 Palmerston Place,	1893
	Sir R. W. Philip, 45 Charlotte Square,	1883
	Dr Heytan, 5 Melville Crescent, *Emeritus*	1874
185	Dr Frederick Porter, 65 Morningside Road,	1904
	Dr W. G. Porter, 10 Manor Place,	1907
	Dr Edmond Price, 13 Alva Street,	1909
	Dr Harry Rains, 16 Great Stuart Street,	1896
	Dr Douglas G. Read, Lauenburg, St Barnabas Road, Cambridge,	1905
190	Dr H. S. Reid, 3 Hampton Terrace,	1910
	Dr A. Graham Renton, 7 Castle Terrace,	1913
	Dr James Ritchie, 22 Charlotte Square,	1873
	Dr James Ritchie, 14 Spottiswoode Gardens,	1907
	Dr W. T. Ritchie, 9 Atholl Place,	1900
195	Dr George M. Robertson, Tipperlinn House, Morningside Place,	1900
	Dr Robert Robertson, 20 Royal Circus,	1901
	Dr William Robertson, 10 Stirling Road,	1902
	Dr W. Ford Robertson, 48 Northumberland Street,	1893
	Dr W. G. Aitchison Robertson, 2 Mayfield Gardens,	1892
200	Dr R. M. Ronaldson, 9 Melville Crescent,	1895
	Dr T. R. Ronaldson, 8 Charlotte Square,	1877
	Dr A. Aitken Ross, 8 Newington Road,	1902
	Dr D. Maxwell Ross, Royal Asylum, Morningside,	1913
	Dr J. Stuart Ross, 15 Lennox Street,	1908
205	Dr William Russell, 3 Walker Street, *Vice-President*,	1884
	Dr F. G. Salt, 50 Gloucester Square,	1895
	Dr James Scott, 43 Manor Street,	1895
	Dr Flemyng Shennan, 95 Mayfield Road,	1897
	Dr W. C. Silver, 2 Lockhart on Gardens,	1900
210	Emeritus Professor Sir Alexander R. Simpson, 52 Queen Street,	1859
	Dr T. D. Simpson, 9 Hampton Terrace,	1905
	Dr G. F. Barbour Simpson, 43 Manor Place,	1900
	Dr John W. Simpson, 19 Lansdowne Crescent,	1901
	Dr Arthur H. H. Sinclair, 3 Charlotte Square,	1909
215	A. A. Scot Skirving, Esq., C.M.G., 8 Randolph Crescent *Secretary*,	1897
	Dr A. T. Sloan, 22 Abercromby Place,	1885
	Dr J. Cormack Smith, 9 Brunton Terrace,	1896
	Dr F. I. Spriggs, Duff House, Banff,	1913
	Dr J. W. Steven, 13 Manor Place,	1900
220	Dr John Stevens, 78 Lelwarth Terrace,	1892
	Dr Norman L. Stevenson, 7 Alva Street,	1902
	Dr Ian Struthers Stewart, 13 Castle Terrace,	1906
	Dr J. S. Stewart, 15 Merchiston Place,	1885
	Dr Robert Stewart, 25 George Square,	1892
225	Dr William Stewart, 140 Terry Road, Leith,	1889
	H. J. Stiles, Esq., 9 Great Stuart Street,	1883
	John W. Strethers, Esq., 2 Chester Street,	1901
	W. J. Stuart, Esq., 9 Chester Street,	1902
	Dr William G. Sym, 12 Alva Street,	1889
230	Dr W. Taylor, 12 Melville Street,	1871
	Dr W. Macrae Taylor, 8 Melville Street,	1903
	Dr C. H. Thatcher, 8 Melville Crescent,	1876
	Dr L. H. L. Thatcher, 8 Melville Crescent,	1910
	Dr R. Thin, 25 Abercromby Place,	1870
235	Professor Alexis Thomson, 30 Drumsheugh Gardens,	1887
	Dr H. Tarrant e Thomson, 3 Hillside Crescent, *Treasurer*,	1900
	Dr John Thomson, 14 Oxford Terrace,	1887
	Dr Thomas James Thomson, 3 Greenhill Park,	1911

		Date of Admission.
	Dr George Thyne, 13 Brunswick Street,	1904
240	Dr T. J. Thyne, 16 Randolph Crescent,	1898
	Dr John Tod, 67 Ferry Road, Leith,	1895
	Dr H. M. Traquair, 30 Walker Street,	1910
	Dr F. J. Turnbull, 6 Randolph Place,	1896
	Dr Dawson Turner, 37 George Square,	1890
245	Dr Logan Turner, 27 Walker Street,	1892
	Henry Wade, Esq., 6 Manor Place,	1903
	Dr Alexander Walker, 1 Lauder Road,	1907
	Dr Norman Walker, 7 Manor Place,	1891
	D. Wallace, Esq , C.M.G., 29 Charlotte Square,	1887
250	Dr Chalmers Watson, 11 Walker Street, *Editor of Transactions*,	1894
	A. Pirie Watson, Esq., 58 Melville Street	1911
	Dr W. de Watteville, Kingussie,	1902
	Dr A. D. Webster, 18 Minto Street,	1883
	Dr H. L. Watson-Wemyss, 7 Coates Gardens,	1911
255	D. P. D. Wilkie, Esq., 56 Manor Place,	1909
	Dr James Wilson, 53 Inverleith Row,	1900
	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
	Dr A. Murray Wood, 180 Ferry Road, Leith,	1907
	Dr Russell E. Wood, 9 Darnaway Street,	1883
260	Professor John Wyllie, 44 Charlotte Square,	1868
	Dr James Young, 2 Randolph Place,	1912

(*b*) Members Exempted under Rule V. from paying the Annual Subscription.

	Dr D. Aitken, 152 *Packington Street, Islington, London, N.*,	1887
	Dr H. de M. Alexander, *Aberdeen District Asylum, Aberdeen*,	1901
	Dr D. H. Anderson, 143 *Abbey Road, Barrow-in-Furness*,	1887
265	Dr John Anderson, *Pitlochry*,	1896
	Dr J. A. Armitage, 58 *Waterloo Road, Wolverhampton*,	1887
	Dr F. L. Atkinson, *Victoria Road, Devonport, Auckland, New Zealand*,	1904
	Dr W. Badger, *Penicuik*,	1882
	Dr Edwin Baily, *Oban*,	1883
270	Dr Andrew Balfour, *Khartoum*,	1895
	Dr J. H. Balfour, *Portobello*,	1881
	Dr Alexander Ballantyne, *Dalkeith*,	1872
	Dr Noel D. Bardswell, *Midhurst Sanitorium, Sussex*,	1901
	Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*,	1890
275	Sir George T. Beatson, K.C.B., 7 *Woodside Crescent, Glasgow*,	1895
	Professor James M. Beattie, M.B., *The University, Sheffield*,	1904
	Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*,	1889
	Dr Charles Begg, 62 *Pulteney Street, Bath*,	1900
	Dr Benjamin D. C. Bell, *Kirkwall*,	1889
280	Dr G. J. H. Bell, *Bengal Army*,	1884
	Dr J. H. Montgomerie Bell, *Brunswick Square, Camberwell*,	1905
	Dr G. H. Bentley, *Kirkliston*,	1877
	Professor Richard J. A. Berry, M.D., *University of Melbourne*,	1893
	Dr J. S. Beveridge, *Melton Constable, Norfolk*,	1861
285	Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*,	1865
	Dr D. G. Braidwood, *Halkirk, Caithness*,	1889
	Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*,	1884

		Date of Admission.
	Dr H. Grey Brown, 47 *Eban Street, Sandhaven*,	1905
	Dr James Murdoch Brown,	1885
290	Dr John Brown,	1878
	Dr J. Macdonald Brown, 2 *Fragnall, Hampstead, London, N.W.*,	1883
	Dr William Brown, *Braemar*,	1904
	Dr Lewis C. Bruce, *The Asylum, Murthly, Perthshire*,	1895
	Dr William Hartley Bunting, 22 *Faington Row, Edgbaston, Birmingham*,	1900
295	Dr Malcolm Campbell, *Irochin*,	1904
	Dr D. A. Carruthers, 5 *Charlotte Street, Perth*,	1901
	Dr S. W. Carruthers, 44 *Central Hill, Norwood, London, S.E.*,	1899
	Professor Cleland, *The University, Glasgow*,	1864
	Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*,	1878
300	Dr William Craig, *Cowesmouth, Ayr*,	1894
	Dr J. R. Cresse, 2 *Oak Terrace, South Shields*,	1885
	Dr J. Francis Crombie, *Norton Lennard*,	1897
	Dr Bampfylde Daniell, *Adderley Street, Cape Town*,	1883
	Dr G. A. Davies, *Musselburgh*,	1900
305	Dr P. M. Deas, *Hunted House, Exeter*,	1868
	Dr Frederick Dittmar, 72 *Murray-on-a Gardens*,	1900
	Dr Alex. Don, *Park House, Nethergate, Dundee*,	1889
	Dr A. Home Douglas, 5 *Avenue Kunste, Nice, France*,	1889
	Dr J. J. Douglas, 42 *Cintra Park, Upper Norwood, London*,	1891
310	Dr W. D. Dow, *Dunfermline*,	1879
	Dr J. K. Drysdale, *Grimsargh-on-Sea*,	1901
	Dr Henry J. Dunbar, 118 *Cathcart Road, Cardiff*,	1906
	Dr C. C. Easterbrook, *Cricklewood House, Dundee*,	1899
	Dr J. W. Eastwood, 18 *Farndon Road, Oxford*,	1871
315	Dr F. A. Elkins, *The Asylum, Leavesden, near King's Langley*,	1893
	Dr R. W. Felkin, 12 *Oxford Gardens, North Kensington, London, W.*,	1885
	Dr A. N. Fell, 11 *Lexden Road, Colchester*,	1907
	Dr Samuel Fergusson, *Dykebar House Asylum*,	1902
	Right Hon. Sir R. E. Finlay, K.C., LL.D., *Middle Temple, London*,	1894
320	Major A. M. Fleming, R.A.M.C., *c/o Grindlay & Co., 54 Parliament Street, London, W.*,	1912
	Dr Charles Forsyth, *Kwantien Lunatic Asylum, Hong Kong*,	1903
	Dr Louise, *South Africa*,	1908
	Dr Simson C. Fowler, *Jumpers Green*,	1892
	Dr I. W. Freeston, 47 *Newhall Street, Birmingham*,	1893
325	Dr Dyce Fraser, 163 *Cornwall Road, London, S.E.*,	1883
	Dr William Gayton, 11 *Redheaton Avenue, North Finchley, London, N.*,	1886
	Dr Campbell Geddes, *Professor of Anatomy, McGill University, Montreal*,	1913
	Dr Rae Gibson,	1910
	Dr T. Paul Gray, 94 *Adelaide Road, Hampstead, N.W.*,	1899
330	Dr W. C. Greig, *Otago, New Zealand*,	1884
	Dr George K. Grimmer, *Canada*,	1887
	Dr Groesbeck, *Cincinnati*,	1875
	Dr W. R. T. Guldan, *Ireland Cross, Devon*,	1893
	Dr Haig, *Gainmore, Crieff, N.B.*,	1912
335	Dr John Hamilton, *Dykebar, Renfrewshire*,	1883
	Dr Archibald Hall, *Montreal*,	1855
	Dr J. W. Harap, *Penn Road, Wolverhampton*,	1887
	Dr John Hardie,	1895
	Dr A. W. Hare, *Newquay, Cornwall*,	1883
340	Dr J. Howe Hay, *Ayr*,	1880
	Professor J. Berty Haycraft, *Llanrhos, Cardiff*,	1884
	Dr Stanley Haynes, *Malvern, Worcestershire*,	1894

		Date of Admission.
	Dr R. Dundas Helm, 13 *Portland Square, Carlisle,* . .	1892
	Dr David Hepburn, Professor of Anatomy, *University College, Cardiff,*	1902
345	Dr R. E. Horsley, *Crail, Fife,*	1886
	Dr James Hunter, *St Catherine's, Linlithgow,* . .	1890
	Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.,*	1887
	Dr David Huskie, *Moffat,*	1899
	Dr Robert Inch, *Gorebridge,*	1887
350	Dr James Johnston, 53 *Princes Square, Bayswater, London, W.,*	1871
	Dr J. Carlyle Johnstone, *The Asylum, Melrose,* . .	1882
	Dr J. Keay, *Bangour Asylum, Uphall,* . . .	1887
	Dr A. J. Keiller, *New Brunswick, Canada,* . . .	1889
	Dr George Keith, *Moidart Cottage, Currie,* . . .	1845
355	Dr Skene Keith, 58 *Upper Berkeley Street, Portman Square, London, W.,*	1885
	Dr W. Scott Lang, ...	1868
	Dr J. W. Leech, 12 *Eldon Square, Newcastle-on-Tyne,* .	1901
	Professor R. F. C. Leith, M.B., *The University, Birmingham,*	1886
	Dr G. R. Livingston, 47 *Castle Street, Dumfries,* . .	1901
360	Dr James Lochead, *Gibraltar,*	1911
	Dr T. Davy Luke, *Peebles,*	1900
	Dr Donald Macaulay, *Jumpers' Gold Mining Co., P.O. Box 1043, Johannesburg,*	1895
	Dr P. M'Bride, *Harrogate,*	1879
	Dr Stuart M'Donald, *Newcastle-on-Tyne,* . . .	1901
365	Dr John A. Macdougall, *Cannes, France,* . . .	1875
	Dr William C. M'Ewan, *Prestonpans,*	1892
	Professor Sir J. M'Fadyean, 101 *Great Russell Street, London, W.C.,*	1888
	Dr F. Reid Mackay, *New Zealand,*	1899
	Dr John Mackay, *Aberfeldy,*	1881
370	Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed,* .	1893
	Professor M'Kendrick, *Maxie Burn, Stonehaven,* . .	1870
	Dr James Mackenzie, 17 *Bentinck Street, London,* .	1894
	Dr Robert Mackenzie, *Napier, Nairn,*	1893
	Dr Theodore C. Mackenzie, *The Asylum, Lochgilphead,* .	1904
375	Dr William A. Mackintosh, 3 *Park Terrace, Stirling,* .	1896
	Dr William MacKirdy, 157 *Lincoln Road, Peterborough,* .	1907
	Dr David W. Maclagan, *care of Dr Harrison, Eltham, Taranaki, New Zealand,*	1901
	Dr J. Shaw M'Laren, *India,*	1887
	Dr Roderick M'Laren, 23 *Portland Square, Carlisle,* .	1882
380	Dr J. C. G. Macnab, *South Africa,*	1904
	Dr Alex. H. Macpherson, ...	1901
	Dr G. Douglas M'Rae, *Glengall House, Ayr,* . . .	1904
	Dr William B. MacTier, 7 *Queen's Gardens, St Andrews,* .	1901
	Dr Samuel Macvie, *Chirnside,*	1897
385	Dr J. W. Martin, *Charterhall, Newbridge, Dumfries,* .	1888
	Dr J. B. Mears, ...	1907
	Dr Andrew W. Messer, *Lemington, Scotswood-on-Tyne,* .	1899
	Dr James Middlemass, *Boro' Asylum, Ryhope, Sunaerland,* .	1895
	Dr E. A. Mills-Roberts, *Bethesda, Bangor,* . . .	1897
390	Dr J. Moolman, *Cape of Good Hope,*	1877
	Dr J. E. Moorhouse, 6 *Melville Terrace, Stirling,* .	1901
	Dr A. E. Morison, *Mowbray Villa, Sunderland,* . .	1889
	Dr J. Rutherford Morison, 14 *Saville Row, Newcastle-on-Tyne,*	1882
	Dr John Mowat, *Caithness,*	1885
395	Professor Robert Muir, *The University, Glasgow,* . .	1894
	Dr D. G. Macleod Munro, 1 *Oriel Villas, Cheltenham,* .	1898
	Dr Peter Murray, *Durie Street, Leven,* . . .	1904

		Date of Admission.
	Dr W. H. Myers, *Indiana, U.S.A.*,	1906
	Dr Leith Napier, *The General Hospital, Adelaide, South Australia*,	1870
400	Dr Ernest F. Neve, *Srinagar, Kashmir, India*,	1884
	Dr Peter Orphoot, *North Berwick*,	1865
	Dr T. Wyld Pairman, *Hinemoa, Moffat*,	1884
	Dr P. Pattison, 4 *Novar Crescent, Kirkcaldy*,	1907
	C. Balfour Paul, Esq., 8 *Chatsworth Square, Carlisle*,	1904
405	Professor Bell Pettigrew, *St Andrews*,	1873
	Dr Alexander Peyer, *Zurich*,	1893
	Dr J. A. Philip, *Rue Victor Hugo, Boulogne-sur-Mer, France*,	1878
	Dr Harvey Pirie, *Nairobi, East Africa*,	1905
	Dr J. H. Pringle, 172 *Bath Street, Glasgow*,	1886
410	Dr S. Hale Pringle, *Bishop's Castle*,	1885
	Dr R. B. Purves, 5 *Minster Yard, Lincoln*,	1899
	Dr J. Maxwell Ross, *St Ruth's, Maxwelltown, Dumfries*,	1882
	Dr J. R. Home Ross, *Burnside*,	1888
	Dr Joseph C. Ross, *Hutchinson*,	1884
415	Dr S. Rumboll, *Cavendish House, Leeds*,	1887
	Dr Thomas Russell, *Beechwood, Parkhead, Glasgow*,	1888
	Dr Gordon Sanders, *Cannes, France*,	1892
	Dr E. A. Saunders, *Grahamstown, South Africa*,	1884
	Dr Thomas R. Scott, *Musselburgh*,	1884
420	Dr W. Vernon Shaw, 42 *Yorkersgate, Malton, Yorks.*,	1905
	Dr Herbert D. Shepherd, 45 *Kedleston Road, Derby*,	1903
	Dr G. E. Shuels, 229 *Geary Street, San Francisco*,	1887
	Dr W. H. Shurtleff, *Port Fairy, Victoria, Australia*,	1883
	Dr William Simmers, *Gordon, Berwickshire*,	1894
425	Dr Herbert Sloman, 65 *Goldington Road, Bedford*,	1906
	Dr John Smith, *Braehead, Kirkcaldy*,	1889
	Dr William Smith, *America*,	1890
	Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia*,	1892
	Dr Van Someren, *Northwold, Middlesex*,	1845
430	Dr J. W. Somerville, 12 *Abbotsford Road, Galashiels*,	1905
	Dr Arthur J. de Spiganovicz, *Lumloe, Thame*,	1895
	Dr A. M. Stalker, 156 *Perth Road, Dundee*,	1893
	Dr Graham Steell, 90 *Mosley Street, Manchester*,	1877
	Professor Stephenson, *University, Aberdeen*,	1861
435	Dr John Stevenson,	1900
	Dr Robert Stirling, 4 *Athol Place, Perth*,	1891
	Professor Ralph Stockman, *The University, Glasgow*,	1891
	Dr H. R. Storer, *Newport, Rhode Island, U.S.A.*,	1855
	Dr John Strachan, *Dollar*,	1867
440	Dr John Struthers, *Nyasaland, Transkei, South Africa*,	1895
	Dr J. T. Sturrock, *Arima, Bromley Park*,	1887
	Professor J. Symington, *Queen's College, Belfast*,	1878
	Dr C. C. Teacher, *North Berwick*,	1887
	Dr C. Templeman, *Sanitary Office, Bank Street, Dundee*,	1891
445	Dr J. A. Douglas Thompson, *Cheetham House, Halesowen, Manchester*,	1901
	Dr J. C. Thomson, *Dumfries*,	1910
	Dr J. Stitt Thomson, *Castle Hill House, Lincoln*,	1877
	Dr J. Batty Tuke, Jr., *Morningside*,	1886
	Dr R. S. Turner, *Kirkcaldy*,	1867
450	Dr T. Ilgar Underhill, *Dunsdon, Burnt Green, Worcestershire*,	1885
	Dr A. R. Urquhart, *Murray House, Perth*,	1893
	Dr Gopal Govind Vaze, *care of H.H. the Rajah of Miraj, Bombay, India*,	1895
	Dr David Waterston, F.R.C.S.E., *London*,	1900
	Dr Walter de Watteville, *Kingsgate*,	1902

		Date of Admission.
455	Dr J. H. Douglas Webster, ...	1907
	Dr A. B. Whitton, *Aberchirder*, ...	1886
	Dr H. Douglas Wilson, 3 *Rippon Road, Harrogate*, . .	1904
	Professor G. Sims Woodhead, *Pathological Laboratory, New Museums, The University, Cambridge*, ...	1883
	Dr Yellowlees, 6 *Albert Gate, Dowanhill, Glasgow*, . .	1862

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TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION XCII., 1913-14

A Special Meeting.—October 29, 1913

Mr J. M. Cotterill *in the Chair*

THE NEED FOR EUGENIC REFORM

By Major Leonard Darwin, President of the Eugenics Education Society

(*Conjoint meeting with the Obstetrical Society to inaugurate the new meeting-place.*)

Linnæus, writing in the middle of the eighteenth century, declared that species are now as numerous as when Providence first created them in the beginning, and that, in accordance with the laws of generation then laid down, the beings originally created produced only forms similar to themselves, with the result that the species now with us are no more numerous than they were at the first. In writing thus this great naturalist was only expressing the belief in the immutability of the different forms of life almost universally held in all civilised countries until the middle of the last century. It is true that the suggestion that the world as we see it now is the outcome of a long series of continuous changes, generally similar in kind to those actually taking place around us to-day, had been made by several philosophers in ancient days; whilst from time to time some more advanced thinker would express the belief that every animal, including man, is the lineal descendant of some more primitive type of living creature. But those who held such views were always so few in number that the general acceptance of their beliefs—the practically universal acknowledgment amongst thinking men during the last fifty or sixty years of the truth of the

principle of evolution can only be described as a complete revolution in thought. But must not the adoption of the belief that by evolution alone can we account for the visible facts of life tend to produce material alterations in the way in which we regard many practical questions? Is not every revolution in thought inevitably accompanied by more or less considerable changes in policy and social customs? The truth is that it is quite impossible for us to go on acting as if we held precisely the same beliefs as our ancestors; and the aim of the eugenist is both to guide and to hasten the reforms in social customs and in legislation which must in any case result from the acceptance of the doctrine of evolution.

But are any new methods of reform necessary besides those which we are now so earnestly striving to introduce, it may be asked? Any one who studies our Poor Law statistics, or who looks at our ever-growing lunatic asylums, homes for imbeciles, and hospitals, or who considers our criminal records, or who merely takes a single walk in our city slums—anyone who ponders over the subjects thus brought to his mind must at all events feel profoundly dissatisfied with the existing social condition of his country. Splendid efforts are no doubt now being made on all sides; but, when we remember how long this noble strife has lasted, we are absolutely driven to inquire whether, in addition to all the good now being done, some other and hitherto untried methods are not also essential to secure the progress of the nation, or indeed to safeguard it from decadence. If, as we hold, it will be necessary to continue in the future as in the past to try to alleviate or mitigate the sufferings of our fellow-creatures by the application of immediate remedies, it will be absolutely encumbent on us henceforth also to recognise the necessity of searching for the ultimate sources of all these evils in the light of our ever-advancing knowledge of the laws of nature. In the past the efforts of the social reformer have been almost entirely directed to conferring immediate practical benefits on man by altering his mental and physical surroundings. Without relaxing these efforts, we must also in the future endeavour to ensure that our descendants shall actually enter the world with a better natural equipment than that which we possess wherewith to fight the battle of life. As long as complete ignorance prevailed as to the way in which the generations of man succeeded each other, it was inevitable that all philanthropic efforts to help the human being should have begun at his birth. But now that this ignorance is being rapidly dissipated we are bound to see if something cannot be accomplished before his

birth ; and this is the aim of the eugenist. We hold that the world is so constituted that all the good and useful inborn qualities of man, as well as all his evil tendencies, have been evolved in accordance with what are called the laws of nature ; and our hope and belief is that by studying those laws, and by utilising the processes thus discovered by which man has been evolved from some lower type of life, we shall now be able to take steps tending to ensure that the man of the future shall be a far nobler being than the man of to-day.

We eugenists are no doubt not yet in a position to formulate a policy with great precision ; and I for one hope that we shall not attempt to do so for a long time to come, because scientific dogmas are apt to be the death of scientific progress. Moreover, when it is remembered that Sir Francis Galton, the founder of our science, wrote his first paper on this subject in the year 1865, and that we have not therefore yet celebrated the jubilee of this movement, no one ought to be surprised that more has not been done and that much remains still unknown. This fog of doubt, which is largely due to our ignorance of the methods adopted by Nature in promoting racial progress, is however certainly slowly clearing away ; and even now through it we believe that we see quite clearly that the selection of the ancestry of the coming generations—the selection of the stock from which the race of the future is to spring—has been at all events one of Nature's main methods of ensuring racial advancement. But if Nature has always as it were paid the closest attention to the factor of heredity in dealing with all animals and plants, and if we wish to see a continuance of the progress thus made in the past, the men of the future becoming healthier, happier, and nobler than the men of to-day because of their superior inborn qualities, surely if this be our aim we clearly see that the duty of studying the question of human heredity is imperatively imposed upon us. In short, whilst we hold that the social reformer should receive every encouragement in his efforts towards the betterment of human surroundings, we eugenists are convinced that it is essential at the same time to adopt certain reforms based on the factor of heredity. Between these two movements—the environmental and the heredity—there need, we think, be no hostility whatever ; for both are absolutely necessary to ensure human progress in all directions. The breeders of race-horses do not waste their time in quarrelling as to which is the more important for their horses, good training or good pedigrees ; and the breeders of mankind may in

large measure leave it an open question which is the more important, environment or heredity. And this is a point which needs emphasising, because misconceptions as to the opinions we hold on these questions have given rise to ill-founded prejudices against us.

The confession which I have just made that the eugenic programme is somewhat indefinite must, however, not be held to imply that there is any lack of work for the eugenist now to do. To convince our fellow-countrymen that human heredity is a subject imperatively demanding our attention; to advocate those moral and legislative reforms which even now we see to be advisable; and to continue those scientific investigations which are necessary to enable us to advance with greater confidence in the future—here in all probability is work enough for all our lifetimes. And firmly believing as I do that all who are willing can play some part in thus helping forward this great social movement, I sincerely trust that a Eugenic Society may be founded in the capital of Scotland, so long the seat both of morality and of science.

My object to-night will merely be to briefly allude to some of the unsolved problems of eugenics, and to indicate some of the ways in which Eugenic Societies can aid both in their solution, and also—and this is perhaps their more important function—in influencing public opinion in the desired directions in the hope of thus promoting the necessary moral and legislative reforms. But before doing so I should like to suggest to those joining this society that it is well to take some little pains to grasp what is really included within the scope of the science it is especially intended to promote. All who sympathise with this movement will probably have read the well-known definition of eugenics finally adopted by Galton, namely, that it includes "the study of the agencies under social control that may improve or impair the racial qualities of future generations, either physically or mentally." Now that definition might at first sight appear, to those who have grasped its full meaning, to be all that is needed both to avoid misconceptions and to attract the attention of the public to this subject. Experience, however, proves that a definition standing alone is almost useless, because, amongst other reasons, the meaning it conveys depends in great measure on the mental atmosphere, so to speak, of the inquirer. This definition has been often wholly misunderstood, whilst perhaps still more frequently it has resulted in the mind being focussed on one only of the many branches of this subject. For example, the words "racial qualities" will forcibly recall to the minds of many

social reformers what have been described as "racial poisons," including alcohol, lead, etc., and may thus lead to the belief that eugenics is mainly concerned with the cure of the undoubted evils arising from such causes. Without going deeply into these vexed questions, a word or two as to how far this view is justifiable may be advantageous.

Though the direct evil effects of alcohol on those who take it in excess, at all events, as well as the moral and material deterioration of their families, are quite unquestionable, yet the eugenist, looking to the nation in the future, must also inquire whether moderate or hard drinking does affect the descendants of the drinker or the drunkard as regards their innate qualities. On this point we are still in doubt, although many confident assertions have been made. There is, it is true, some evidence to show that the sons and daughters of a drunkard are thus damaged as regards their inborn qualities; but, if we look to the actual biological effects on the third and subsequent generations, we are, in my opinion, in complete ignorance. Moreover, as regards those dread contagious diseases which produce such certain and terrible effects on the children of the diseased, here again the biological consequences to future generations are quite problematical. It seems then that the damage done both to the current generation and to their unborn children alone affords ample justification for a campaign against drink and disease, a campaign in which the eugenist should be all the more willing to join because it may yet be proved that these "racial poisons" do injuriously affect posterity for an indefinite number of generations, in which case they would be shown to be eugenic factors of prime importance. We must not, however, neglect the other possible alternative, and we ought to admit that, however vast would be the immediate benefits resulting from the abolition or destruction of racial poisons, yet it may well be that this would not be a step tending to produce a progressive advance in the inborn characters of mankind, which is the true aim of the eugenist. If this be so, surely it follows that, without relaxing our efforts in this noble social campaign, we yet should not remain satisfied until we have ascertained whether some steps cannot be taken which would unquestionably produce that most desirable result.

All around us we see efforts being made to improve the environment of the people, efforts with which I have already expressed my sympathy in the most emphatic manner. But with reference to reforms of this type, if we look the facts of life in the face, we must

acknowledge that as regards all changes in physical conditions, such as better housing and sanitation, we cannot tell how long the improvements now being made will last, nor do we know whether they will tend automatically to promote any corresponding advance in the inborn qualities of the race. Even if we do not join in the vehement denial of the inheritance of acquired characters, now the attitude commonly adopted in the world of science, yet no one can in truth pretend to estimate the eugenic value, or the value to the generations of the more distant future, of any purely environmental reform. In order that we may stand on firmer ground we eugenists must continue to urge that further inquiry be made as to the biological effects of changes in human surroundings, an inquiry of the greatest difficulty. And, pending the result of such researches, we must continue to point out that if our object be to ensure the continued progress of the race, and if we wish to make certain that we are taking some steps in this direction, we must not confine our attention to reforms affecting environment. But where then are we to look if this be our quest? Is it not obviously to the factor of heredity, by means of which the inborn qualities of one generation are passed on to the next, to which we ought mainly to look when taking thought concerning the racial advancement of mankind? In fact we have by a somewhat different route again reached the conclusion that heredity is the key by means of which the door leading to the future advancement of mankind must be unlocked.

But even if this view be adopted we have many difficulties to face before we can decide on the practical steps which should now be taken. Let us begin by imagining mankind first arranged in order according to the qualities held to be desirable, and then divided into two equal groups, when there would be one group composed of individuals all above, and another of those all below the average in what Galton called civic worth. This imaginary division having been made, it is no doubt evident that the main aim of the eugenist should be to decrease rate of multiplication in the lower of these two halves of humanity relatively to the rate of multiplication in the upper half; for the greater the success of such efforts, the greater would be the proportion of our descendants who would have sprung from superior stocks, and the more certainly would progress be thus ensured through the agency of heredity. But before the best methods of securing these results can be ascertained, many problems have to be solved, and much hard work must be undertaken. The first question which naturally arouses our atten-

tion is whether as a fact our nation is now advancing as regards its inborn qualities, or whether we are degenerating; that is whether the less fit are multiplying so fast that they are, as it were, swamping the more fit, or whether, the reverse being the case, we are still on the upward path of racial progress. In order to decide in which half of humanity the rate of multiplication is highest, we must obviously have some method of sorting human beings into groups according to their innate qualities, or their biological fitness; and this we have now no means of doing. We can, it is true, generally say whether one group is richer or poorer than another; but does this afford any indication of their relative fitness? Or, in other words, is there any correlation between wealth and innate ability? Here is a problem urgently demanding our attention, to which but little thought has yet been given, and concerning which there is still much doubt. As a single example of the way in which an answer to this question may be sought, what is being done by my society in this direction is perhaps worth mentioning. A scheme has been elaborated by means of which a record will, it is hoped, be obtained of the social classes, or the kind of surroundings from which certain boy and girl emigrants are drawn; whilst subsequently efforts will be made to follow their careers in the colonies. These young people go out without parents, and all under much the same conditions, the element of environment being thus more or less completely eliminated; and their innate qualities can therefore be judged by their subsequent success or failure in life. In this way we ought, therefore, in time to be able to ascertain whether these innate qualities do vary according to the social status of the emigrant's family.

If anyone can suggest any other and more ready method of ascertaining the inborn qualities of the different sections of the nation, we shall indeed be grateful; for, as it is, we have to rely mainly on *a priori* reasoning in considering whether there is any correlation between wealth and innate qualities. Certainly no one can be so foolish as to deny that there are amongst the poor many individuals both very virtuous and very capable, or that amongst the rich many most undesirable specimens are found, by whatever test they may be judged. But it is equally foolish to blind our eyes to the fact that a sifting process is in fact continually in operation; for the naturally strong and able amongst the poor often acquire greater wealth, whilst many of naturally deficient stock amongst the richer classes keep slowly sinking from their more

comfortable estate. In fact it can hardly be doubted that the poorest strata of society do contain an exceptionally large proportion of the biologically unfit, a fact which must fill us with grave alarm when we remember that statistical inquiries prove conclusively that this same section of the community has also an exceptionally high birth-rate. For if the unfit are multiplying more rapidly than the fit, this we are certain must inevitably lead to national degeneracy.

But if the relatively low rate of multiplication of the better-off classes—I am not thinking about the very rich—is a source of danger to the nation, ought we not to consider carefully all possible causes which may account for this alarming fact? If we put aside the possible existence of any direct biological cause for the low fertility of the more cultivated classes, we yet have to inquire whether there are not certain factors acting in this direction in a more indirect manner. Almost every one is considerably influenced by fears concerning the possibility of a diminution in his own comfort in the future, and also concerning the prosperity of any children he has or may have; and such fears inevitably tend to a postponement of marriage and a diminution in the size of the family. Moreover, such causes obviously affect the well-to-do more than the destitute; because the fear of a fall varies with the depth of the possible drop, those already at the bottom being troubled with no such apprehensions. The man in comfortable circumstances dreads the possibility of his children falling in the social scale, whilst no such thought can enter the head of the pauper. If this be the main cause of the comparative infertility of the more prosperous and presumably, on the average, abler classes, we have next to consider how this national disease may be cured.

In this quest our aim must obviously be amongst other things to make respectable parents with families look to their future with greater assurance than they at present feel. Possibly something might be done to increase the now somewhat slender confidence which is felt in regard to the contributions receivable from children in old age, with the result that the production of a family would seem to be less economically undesirable. Then again it would unquestionably be possible to make the income-tax vary substantially according to the size of the family, and to deal with death-duties in a similar way. Another proposal which ought, I think, to be adopted in this country, is that which has actually been tried at Schoneberg, near Berlin, namely the adoption of a scale of pay for

all government employés, dependent in certain measure on the number of their children ; for, where none but good workmen are employed, this system would unquestionably be eugenic in its tendencies. In fact there are several reforms to which the politician can devote his energies with confidence as to their beneficial eugenic results.

The expedients just mentioned for lightening the financial burdens of parenthood would, no doubt, touch but a small percentage of the nation, and more drastic remedies applicable to nearly the whole population are certain to be suggested. For example, a bill has been passed by the House of Representatives of Australia with the object of conferring a bonus of £5 to the mother of every child of white race at its birth. All such proposals must, however, be scrutinised with great care before they can be endorsed as regards their eugenic effects ; for any system designed to increase the birth-rate generally may have the effect of increasing it more in the lower than in the upper half of humanity. If any proposed stimulus would be most felt by the least provident classes, as is probably the case with the Australian example just quoted, the results would almost certainly be dysgenic and not eugenic. In short it seems that in our campaign we must not rely too much on such economic stimulants, for our main hope must rest on the possibility of changing the whole mental attitude of the nation in regard to the duties which it owes to posterity.

If the explanation of the low fertility of the well-to-do classes is that they pay too much regard to their future economic position, it may with equal truth be said that the high fertility of the destitute results from many of them taking but little thought of their future welfare ; for innate mental incapacity for forethought often leads to early marriages, large families, and poverty. Although something may be done to mitigate these joint-evils by moral persuasion, yet it must be admitted that the prospects of beneficial results thus being obtained in the very lowest strata of society are but very slight. The man who is not in the least deterred from an improvident marriage by the prospect of immediate want is little likely to be open to eugenic or any other argument. A large class in fact certainly exists which cannot be touched by any such means, and it is within this class that the vast output of undesirable citizens is taking place which the eugenist is striving to lessen. How can this be done ? Possibly some steps might and should be taken in the direction of making those who indulge in reckless marriages feel

more immediately the unpleasant economic consequences thus arising; and this would be perfectly legitimate, considering that by such marriages they inevitably throw a heavy burden on the shoulders of their more prudent neighbours in the form of increased poor rates, etc. But if we look the facts of life fearlessly in the face we are driven to the conclusion that, for a large section of the community, the only methods by which the desired lessening of fertility can be obtained are sterilisation and segregation.

As regards sterilisation, I do not propose to discuss this subject to-night at length. In fact I merely wish to express the opinion that whilst, on the one hand, this question should be approached with the utmost caution and in a most conservative spirit, yet, on the other hand, it would be well to watch carefully what is taking place in America. In eight States of the Union, laws have been passed authorising sterilisation in the case of certain classes of degenerates; and, if little has as yet been done under cover of these laws, this may be due to a doubt as to whether they are constitutional, and therefore valid.

As regards segregation, this method of dealing with undoubted degenerates must now be regarded as well within the region of practical politics; for the eugenic arguments in favour of the Mental Deficiency Bill, though they aroused strong opposition, yet unquestionably carried much weight in many quarters. If we accept the explanation of the high fertility of the unfit already given—namely, that it is due to a certain deficiency on their part in mental powers—we should expect to find that families amongst the definitely feeble in mind are exceptionally large; and this is undoubtedly the case. The Eugenics Education Society had, therefore, no hesitation whatever in placing the Mental Deficiency Bill in the forefront of their programme, and in promoting it by every available means. This they did by endeavouring to influence Members of Parliament in the Constituencies, by appearing before private Committees in the House of Commons, by speaking at meetings, and by circularising the local authorities throughout the country. The victory has now been won, the Bill having passed; but more work of this kind may at any moment become urgently necessary, work in which local Eugenic Societies could play a most useful part.

Besides semi-political agitation of this kind, serious scientific work is also needed in reference to the mentally defective. We

want many more pedigrees of families containing feeble-minded individuals; and these must be collected with all the many precautions needed in such cases. These records are required in order not only to ascertain with greater accuracy the degree of probability of feeble-minded children appearing under given family conditions, but also to solve certain mendelian problems. For example, is feeble-mindedness a simple recessive character? The asserted fact that when two feeble-minded persons marry, *all* their children are feeble-minded, points to this conclusion; whilst other facts are said to point to a different view. In short, much still remains to be ascertained with regard to many genetic questions.

Eugenists ought, in fact, not to remain satisfied for long either as regards scientific inquiry or practical legislation with dealing with the feeble-minded alone; for there are many other classes of degenerates, notably criminals and paupers, whose multiplication ought to be diminished. In both these classes the trouble is we believe closely correlated with inborn mental defects, and more pedigree work is greatly needed in order to enable us to speak with greater confidence concerning the degree of probability of these characteristics being inherited.

Moreover, the whole question of psychological tests demands further attention. It is true that the Binet System has been carefully elaborated in France, by means of which it is held that criminals can be graded according to their mental capacity; whilst Prof. Münsterberg, in his "Psychology and Crime," urges the value of measuring by definite experiment various physical attributes, such as accuracy of perception of touch, in connection with mental degeneracy. In fact what we want to be able to do, as it seems to me, is to be able to mark out as a class apart those individuals who have been proved to be undesirable or defective as judged by *all* the three following tests:—

(1) In the case of criminals, by having been convicted of several crimes, the number depending on their gravity; and, in the case of paupers, by having been frequently in and out of the workhouse.

(2) By having been proved, either by evidence or by definite physical or psychological tests, to be either physically or mentally defective; and,

(3) By having been reported by the prison or workhouse authorities, as the case may be, to be unable or clearly unwilling to gain an honest livelihood.

As regards criminals thus judged to be degenerates, they should

be dealt with, not as needing punishment, but either as merely requiring either protection against themselves, or control for the purpose of safeguarding the public from present injury or future hereditary taint; that is to say that they should be treated with great consideration, provided that their periods of confinement are never brief. As regards paupers coming within these three categories, much the same remarks are applicable; and, for their detention, labour colonies ought to be established in the United Kingdom in pursuance of the example set to us by Belgium and Switzerland. Neither with criminals, nor with paupers, should such detention in the first instance be regarded as being necessarily for life; for those whose failure can fairly be attributed in large measure to misfortune ought to have a chance of regaining their freedom in time. Even if a mistake is sometimes made in letting out a veritable degenerate, the evil will probably soon be remedied by his return to prison or to the workhouse. Such systems, in fact, must be judged by their results taken as a whole; and, as regards this proposal, it certainly would have the effect of materially reducing the output of defectives, which is all we can hope to do at present.

Eugenics is therefore, as we have seen, founded on the results of scientific investigation; and, in strengthening its foundations, much remains to be done. But since foundations standing alone are useless, what we have to do is to erect on them a moral superstructure; and here, I am convinced, our greatest difficulties will be felt. Our main object must be to sow broadcast throughout the nation, a deep and impelling sense of our obligations towards posterity, a task which may be facilitated in many different ways. Eugenic Education Societies may be founded in various localities, a step which would prove, I believe, to be most beneficial: for every one who, by joining a Society, enrolls himself as one of our army, is quite unconsciously thus induced to become an advocate in our favour in general conversation. Moreover, in definite propaganda work such local help is almost indispensable. For example, at Liverpool certain members of the council of the local Eugenic Society have done excellent service in themselves giving elementary lectures in Eugenics at various institutions, such as workmen's clubs, teachers' associations, etc. Again, such help is needed in making the best use of the local press. Lastly, especial efforts should be made to interest teachers in schools of all kinds in our cause; for, if the eugenic ideal could be properly planted in the minds of all children,

not only would the effect be immediately ennobling, but good fruit would in time grow of itself out of this seed in the attitude adopted towards eugenic problems by the men and women of the future.

In conclusion, what I wish to urge most strongly is that it is not only the help of the scientist and the moralist that we need; for we appeal to every class of the community to join us in our fight against national degeneracy. If only it were more widely known—and here the economist could help us—how enormous is the burden now thrown on the community by the presence in our ranks of vast numbers of degenerates, then we should be better able to show how urgent is the need for reform. Increased taxation, often hidden in the form of increased rent; lowered wages; increased prices; diminished aid in times of trouble; and lessened power of competition in times of peace and of defence in times of war—these are the heavy penalties we are now paying for the ignorance and consequent carelessness of our ancestors with regard to the purity of our race. Are we to be equally careless as regards the future, and shall we pass on this taint with undiminished intensity to posterity? If ever we can succeed both in making the facts on which our creed is built more widely known, and in establishing as an accepted truth our belief as to the grave danger of rapid national degeneracy, then I am certain we shall only have to appeal to our fellow-countrymen in the name of patriotism in order to attract sufficient recruits to our ranks to make the success of our cause a certainty.

Meeting I.—November 5, 1913

Mr J. M. Cotterill, *President, in the Chair*

I. Election of Office-Bearers

The following gentlemen were elected office-bearers:—*President*, Dr John Playfair; *Vice-Presidents*, Dr William Russell, Mr J. W. B. Hodsdon, Dr Melville Dunlop; *Council*, Dr H. A. Leebody, Dr H. G. Langwill, Dr John Eason, Mr Alexander Miles, Mr J. M. Cotterill, Dr Edward Armour, Dr James Wilson, Dr J. Stewart Fowler; *Treasurer*, Dr H. Torrance Thomson; *Secretaries*, Mr A. A. Scot Skirving, Dr Edwin Bramwell; *Editor of Transactions*, Dr Chalmers Watson.

II. Treasurer's Annual Statement

Edinburgh Medico-Chirurgical Society's Accounts

FOR SESSION 1912-1913.

INCOME		EXPENDITURE	
Entrance Fees	£4 4 0	Turnbull & Spears, printing Transactions, Vol. XXXI., Postage, etc.	£68 2 8
Annual Subscriptions	120 10 0	Alex. Frazer, Epidiascope, one-third share	44 0 2
Annual Subscriptions, advance	1 0 0	Alex. Frazer, Microscopes, etc.	5 2 0
Dividends Caledonian Railway Stock	13 3 8	A. & D. Falcon, Stationery	1 3 4
Dividends Dominion of Canada Stock	9 17 9	Edinburgh Co-operative Printing Co.	0 10 0
Interest on Deposit-Receipts	3 7 2	Neill & Co., Billets and Postage	28 17 0
		Post Office Directory	0 7 6
		M'Callum, Teas	5 6 8
		Reporting Meetings	9 0 0
		Rent of Hall	10 0 0
		Session at Hall	1 6 0
		Collector	4 10 0
		James Thin, Bookseller	0 8 6
		Hooker Memorial Fund	1 1 0
		Gardner Bros.	2 9
		Petty Cash	0 18 0
	£152 2 7		£181 4 7

Balance in Bank, November 1912	108 14 7	Balance in Bank, November 1913	79 12 7
	£260 17 2		£260 17 2

STATE OF FUNDS

At November 1912.		At November 1913.	
£350 Caledonian Railway 4% Debenture Stock (Purchase Price)	£484 11 6	£350 Caledonian Railway 4% Debenture Stock (Purchase Price)	£484 11 6
£300 Dominion of Canada 3½% Debenture Stock (Purchase Price)	298 2 6	£300 Dominion of Canada 3½% Debenture Stock (Purchase Price)	298 2 6
Balance in Bank	108 14 7	Balance in Bank	79 12 7
	£891 8 7		£862 6 7

H. TOD LANCE THOMSON, *Treasurer.*

We have examined the accounts and vouchers with which they were compared, and have found them correct.

GEORGE THYNE
JAMES SMITH

Nov. 20th, 1913.

III. Valedictory Address

By the President, Mr J. M. Cotterill.

Gentlemen,—Since the earliest days of this Society, it has been the custom for the retiring President to offer to the Society some remarks on the conduct of the business during his term of office, or to deliver a valedictory address dealing with some subject of professional interest. In some cases these addresses have attained a high level of literary excellence, in others they have been simply a record of work done and progress made by the Society.

My first and most obvious duty, Gentlemen, at the close of my term of office, is to thank you once again most sincerely for the great honour you conferred upon me in electing me President of the Society, and for the great consideration and kindness you have shown me in my conduct of the duties of the Chair.

During my term of office I think we may congratulate ourselves that the Society has continued to flourish, and that a great deal of useful work has been done.

The numbers of the Society are keeping up, the meetings have been well attended, many important and interesting discussions have been held, and in all respects the Society shows signs of continued vitality and prosperity.

Among the many interesting Papers read and discussed were the following :—

" On Perforated Duodenal Ulcer," by Mr Struthers

" A Febrile Epidemic with rapidly Fatal Cases," by Drs M'Neil and M'Gowan.

" On Bone Tuberculosis," by Mr John Fraser.

" On Multiple Neuromata of the Central Nervous System," by the late Dr Alexander Bruce and Dr Dawson.

" On Conduction Defects in the Heart," by Dr Mathewson.

" Trauma as a Factor in Disease," by Dr Alex. James.

The following Discussions were held :—

" On Malingering," introduced by Dr Byrom Bramwell.

" On the Treatment of Simple Fractures," by Mr Cathcart.

" On Vaccine Therapy," introduced by Dr James Ritchie.

" On Intestinal Toxæmia," introduced by Dr William Russell and Mr Alexis Thomson—

and lately we have had an interesting discussion on " Eugenics." This somewhat thorny subject was introduced by Major Leonard

Darwin, to whom our best thanks are due for his kindness in coming to address us, and the interesting and suggestive way in which he dealt with his subject.

While persistent efforts have been made by the Council to bring subjects for discussion before the Society which will elicit a widespread interest amongst the members, it has also been thought right to modify somewhat the method of conducting the clinical meetings, with a view of affording the members a better opportunity than heretofore of personally examining the cases brought forward. This new method is still on trial, and may perhaps with advantage be still further modified.

During my term of office two other important steps have been taken. We have left our old quarters in George Street which were in many respects unsuitable for our purposes, and with the kind approval and assistance of Sir Robert Philip and his Committee, we have moved into these more suitable rooms in Lauriston, where doubtless, when a few additions and alterations have been made for our convenience, we shall find ourselves much better off. Not only is the hall better suited for our numbers but the side-rooms for patients and other purposes are far more convenient.

We have also lately determined to purchase an Epidiascope for the use of the Society, and in this venture we have been joined by the Obstetrical Society and by Sir Robert Philip and his Committee—

The Obstetrical Society propose in future to hold their meetings in this Hall, and we hope that the recent joint meeting of the Medico-Chirurgical Society and the Obstetrical Society, which was that occasion when Major Darwin addressed us, may be the first of several such joint-meetings, where topics of mutual interest to both Societies may be discussed.

I need scarcely remind you that our Society is one of the foremost factors in medical education in Edinburgh, and takes a living interest in all questions pertaining to that subject. We therefore view with great satisfaction the two recent important advances that have been made in medical education in the Edinburgh School. I refer to the recent appointments of Dr William Russell as Professor of Clinical Medicine, and of Dr James Ritchie as Professor of Bacteriology. We are convinced that these appointments will be of the greatest possible value to the School, and that the gentlemen who have been chosen to fill them will bring great honour

to themselves and to their University. May I offer them, in the name of the Society, our very warm congratulations ?

During the last two years the Society has lost several notable members by death.

Professor J. Lucas-Championnière of Paris, a foreign corresponding member, was one of the leading Continental surgeons, and was well known in many departments of surgery, particularly in connection with the treatment of fractures.

Sir John Batty Tuke, President of this Society in 1897, late Member of Parliament for the University during several years, was a member of the Medical Council, and one who distinguished himself by much useful work upon questions affecting medical education. He was a leading authority in mental diseases. A good sportsman and a loyal friend, he will be much missed by a large circle.

Last, and greatest of all, Lord Lister—the apostle of antisepsis —the greatest and most scientific surgeon that ever lived—one of the greatest benefactors of the human race—the most shining monument of our noble profession.

In conclusion, Gentlemen, I beg to thank you most sincerely for your kindly attention, and to congratulate the Society on having procured as my successor in the Chair such a very fitting and appropriate man as Dr John Playfair.

Dr Playfair's excellent business capacity and kindly nature will no doubt render his occupancy of the Chair a great success, and I congratulate him on the great honour which you have conferred upon him by electing him as your President.

Gentlemen, I bid you farewell.

Dr John Playfair, on taking the Chair, thanked the Society most cordially and sincerely for the honour they had done him in electing him President. He felt very unworthy to fill the post, and it was only at the request of the Council that he allowed himself to be nominated. Mr Cotterill in his very able and interesting address alluded to the times through which the Medical Profession had been passing, and when it was represented to him (Dr Playfair) that he might be of some use in smoothing over and trying to remove some of the difficulties which had arisen, he gave way, knowing that he had at the same time the loyal support of the Council and of the individual members of the Society. Further, he had to make an apology. A very tempting chance had come in his way of going to India for the next three months. He would therefore be absent

from the next two or three meetings, and for that he sincerely apologised. He knew, however, the affairs of the Society would be in much more able hands, and he only made the statement in order to show that his absence was not because he had not the interests of the Society at heart. He would do all he could to fill the honourable position to which they had so kindly elected him to the best of his ability.

Dr Byrom Bramwell, in proposing a vote of thanks to the retiring President, said the Society had been exceedingly fortunate during the past two years in having such a distinguished and able President. The success of their Meetings had been very largely due to Mr Cotterill and to the kindly, genial, and admirable way in which he had presided. In his address, Mr Cotterill suggested that each member of the Society should try and influence men who are not members to join. He (Dr Bramwell) fully endorsed that, but went even further: he thought what was wanted was a larger attendance at the Meetings of men who are members: if each member would make it his business to persuade his fellow-members to attend, it would be a very great advantage. The Meetings were not always so well attended as they ought to be in an important Society such as the Medico-Chirurgical. He endorsed what Dr Mathews Duncan said, "that he never had attended a Meeting of this Society without learning something. That was the greatest testimonial that could be given to the value of their Meetings.

Mr Cotterill also referred to the teaching arrangements in the Infirmary. Dr Bramwell cordially agreed with him that these arrangements were admirable so far as they went, but it seemed to him that they were only a stepping-stone to further alterations which would be of still greater importance. The fact was that the students in Edinburgh had not sufficient time to devote to hospital and clinical work: he was certain that until the systematic lectures were cut down and more time devoted to work in hospital they would be competing under very great disadvantages with the large provincial hospitals, in some of which, at all events, the whole morning was devoted to clinical work. He concluded by again thanking Mr Cotterill in the name of the Society for his address, and expressed the hope that it would be published.

Sir Thomas Clouston, in seconding the vote of thanks, said they all agreed most heartily with what Dr Bramwell had said. They had

all been most interested in the address; Mr Cotterill had just hit the proper nails on the head, and anyone who had had to do a similar duty knew that was not an easy thing.

Dr Playfair conveyed to Mr Cotterill the very warm thanks of the Society, and remarked that it made him feel what a difficult task he had before him.

Mr Cotterill thanked the Society most sincerely for the kind things which had been said and for the way in which his efforts had been received. He was truly sorry that his address was not more worthy of the occasion; he hoped they would overlook any deficiencies it had—he trusted its length was not one of them—which were quite made up for by the remarks which fell from Dr Bramwell.

IV. Original Communications

1. THE SUPRARENAL GLANDS: THEIR COMPARATIVE ANATOMY, PHYSIOLOGY, PATHOLOGY, AND RELATION TO CLINICAL MEDICINE

By Ninian Bruce, M.D., M.R.C.P.,
Assistant-Professor of Physiology, University of Edinburgh; Senior Clinical Tutor, Royal Infirmary, Edinburgh

The suprarenal glands in man consist of two portions, a *cortex* and a *medulla*. In the higher mammals the medulla lies in the centre of the gland and is surrounded by the cortex. If, however, we trace the suprarenal glands through the vertebrate series, we find that in the fishes the cortex and the medulla form two separate organs which do not appear to be related to each other in any way. The cortex, in the fishes, is represented by one or more glands lying between the kidneys and known as the "inter-renal body," while the medulla is represented by a whole series of small paired bodies situated on each side of the vertebral column on the branches of the aorta in close relationship to the ganglia of the sympathetic chain and known as the "chromaphil bodies" or "paired bodies." As we ascend the series, we find that in the amphibians the cortex and medulla become associated with each other so as to form one gland which on section shows two kinds of structures, the greater part consisting of cell columns which interlace in all directions and correspond with the cortical portion, while between and among these masses of cells, islets are seen analogous in structure to the paired

chromophil bodies of the fishes and to the medulla of the human suprarenal. In the reptiles the cortical and medullary portions tend to form separate groups, while in the birds the medullary and cortical portions tend to interlace, the former occupying the meshes of the latter, and it is not until mammals appear in the series that a true cortex and a true medulla become differentiated, the former as a rule completely surrounding the latter. In this paper I am only concerned with the medulla of the supra-renal glands.

The medulla of the suprarenals is formed of a network or sponge-work of cell columns which bound venous sinuses. The cells themselves are in close relationship to the endothelium lining these blood sinuses, and in man are mostly of an irregularly polyhedral shape. Although small granules are present in their protoplasm, their secretion, adrenalin, appears to be formed in a fluid form and passes directly into the blood stream.

Our first exact knowledge of the function of the medulla of the suprarenal glands was obtained from the experiments of Schäfer and Oliver, who made an extract from the gland and injected a little of it into the jugular vein of a cat. They found that if a small quantity of such an extract be injected intravenously an immediate rise of blood pressure resulted. This rise occurs immediately after the injection, is of very short duration, and passes off very quickly. If the dose be repeated the same result is again obtained. They also showed that this reaction is the result of a direct action of the extractive substance upon the arteries themselves. Hardly any other agent is capable of producing such an enormous increase of blood-pressure as is obtained after intravenous injection of adrenalin, and what is still more remarkable is the minuteness of the dose which is necessary to produce these results—a fact which is well shown by Schäfer, who points out that the active principle of the suprarenal capsules, administered in the proportion of not more than one-millionth part of a grm. per kilo. of body weight—less than $\frac{1}{1000}$ of a grain for an adult man—is still sufficient to produce distinct physiological effects on the heart and arteries.

Experimental removal of the suprarenal glands in animals results (see later) in death in from 12 to 48 hours, and their destruction in man from any cause, as in Addison's disease, is also fatal.

We thus see that the suprarenal glands, although small in size, are of great importance to the animal economy; they elaborate an internal secretion which passes into the blood and which is an

important element in the maintenance of the tone of the cardio-vascular system.

The development of the suprarenal medulla has been very carefully worked out by Kohn, who showed that it is really part of the sympathetic nervous system. Kohn found that at an early stage in the development of the sympathetic ganglia, when the different portions of the embryonic sympathetic nervous tissue is becoming differentiated into separate ganglia to form the great chain of the sympathetic, a curious change takes place in the cells themselves. They become divided into two quite separate groups, each of which undergoes a separate course of development. The one group of cells, considerably the larger, undergoes a gradual and progressive development into the nerve-cells of the sympathetic ganglia. The other group takes an entirely different course: the cells suddenly increase in size at a rapid rate and develop into peculiar cells which stain brown with solutions of potassium bichromate. Cells which possess this property are known as " chromaffin " or " chromaphil " cells, and no other cells possess this particular staining reaction with bichromate. They form, indeed, a separate tissue-system, the importance of which will be realised when it is understood that only cells containing adrenalin possess this particular reaction. These chromaffin cells are at an early stage indistinguishable from the cells which later become sympathetic ganglion cells, the relation between them having been compared to that of a brother and sister. As development proceeds, large numbers of these chromaffin cells disappear, but a number remain and may be seen if properly examined for in the various sympathetic ganglia, especially the semilunar, in groups of four or five or even more. Although so many of these chromaffin cells are so intimately related to the sympathetic ganglia, the larger groups tend to become separated from them and to appear as isolated masses upon the ventral surface of the aorta. Such groups are very plentiful during fœtal life, but tend to disappear during post-embryonic life. Certain of the largest groups, however, remain permanently, the largest mass persisting as the medulla of the suprarenal glands, while the smaller masses, when they persist, are known as " accessory suprarenal glands " (a term not strictly correct, as they do not consist of both cortex and medulla). The carotid body which lies at the bifurcation of the common carotid artery is a name given to a similar mass of tissue in the neck. We are thus led to the extremely important conclusion that the cells which compose the medulla of the suprarenal glands are really part

of the sympathetic nervous system. If these cells become exhausted of their adrenalin, then they no longer stain yellow with bichromate solutions, and this reaction gives us an exact and much simpler method of examining the suprarenal glands in animals and in man for the presence of adrenalin than the blood-pressure method, it being merely necessary to place the glands, when fresh, in bichromate solution and to cut them later in gum.

My attention was specially directed to this subject by coming in contact with several somewhat acute cases of Addison's disease. It occurred to me that possibly acute Addison's disease might be a far commoner clinical condition than was usually recognised, a fact which could easily be accounted for, as an acute case might easily be overlooked on account of the absence of pigmentation. The fact that death from diphtheritic toxæmia is always associated with swelling of, and hæmorrhage into, the suprarenal glands suggested a line of research, and as the result of a large number of experiments, both of the bichromate and blood-pressure methods, I was able to establish the fact that death from diphtheritic toxæmia in the guinea-pig is associated with intense congestion and swelling of the suprarenals, which have become exhausted of their usual supply of adrenalin, or at least this supply is greatly diminished. By further experiments I established the fact that there is no interaction between adrenalin and diphtheria toxin, so that this exhaustion of the suprarenal glands is not the result of any destruction of the adrenalin itself. It then occurred to me that possibly the secreting cells of the suprarenal glands, being very highly specialised, might possibly be early thrown out of working order by a toxin and unable to manufacture any further supply of adrenalin once the original supply had been all used up, but this was also found not to be the case. (At this stage it may be pointed out that the exhaustion of the suprarenal glands in the rabbit and cat in diphtheritic toxæmia was not found to be so great as in the guinea-pig.) It then occurred to me to test if this reaction were specific for diphtheria, and if perhaps it might not be possible to exhaust the suprarenal glands by other toxæmias. This I found could be done, and I also found that many other reagents, *e.g.* ricin, could also produce a similar result. Later I found any condition which places a great strain upon the organism could produce suprarenal insufficiency and finally suprarenal exhaustion, and that while certain substances are able to cause this exhaustion more markedly than others, *e.g.* diphtheria toxin, it is not produced so much by the toxin as a toxin but rather by the

fact that the toxin throws a certain strain upon the organism which causes an exhaustion of the suprarenal glands *secondarily*.

The next point is to determine the exact mechanism by which this exhaustion is produced. If the abdominal viscera be all carefully removed in a cat (a spinal animal being particularly satisfactory for this experiment) and the splanchnic nerves stimulated, an immediate rise of blood-pressure will be found to take place, proving thus that the suprarenal glands are under the control of the sympathetic nervous system. If this stimulation be continued for some time it is possible to ultimately exhaust the glands.

That exhaustion of the suprarenal glands is a grave condition is shown by the fact that removal of the suprarenal glands is always fatal. Possibly in some cases there may be sufficient accessory suprarenal medullary tissue to replace the loss of the suprarenal glands themselves but never for any great length of time, and this fatal result is due to the loss of their internal secretion, the adrenalin. We have seen that adrenalin, when injected intravenously, has a very marked action on the blood-pressure, a reaction which rapidly takes place and is of only very short duration. But the injection of a large dose of adrenalin suddenly intravenously is purely an artificial condition. A more natural state of affairs may be produced by giving adrenalin subcutaneously (when it is slowly absorbed), or better still by using it in very dilute solution, when it can be run slowly into the jugular vein. With this latter procedure a very dilute solution may be passed slowly but continuously into a vein, and it will be found that it is possible to regulate the blood-pressure to a nicety. If the rate of inflow be accelerated so that a little more adrenalin passes in in a given time, it will be seen that the blood-pressure immediately rises, and if the rate of inflow be diminished the pressure at once falls, the condition resembling much more closely that found normally in the suprarenal gland itself, where a very small quantity of adrenalin is passing continuously into the blood-stream.

The exact point upon which adrenalin acts has been carefully investigated by Elliot, who showed that the action is neither on the sympathetic nerves themselves nor on the muscle wall of the arteries, but on the myo-neural junctions. This myo-neural junction is derived from the muscle and not from the nerve fibre. Every organ which receives its nerve-supply from the autonomic nervous system is supplied by both motor and inhibitory fibres, the one, however, predominating over the other. If the organ be stimulated

by excitation of these nerves, a reaction is found to occur, a reaction which also takes place whenever adrenalin is injected. All organs supplied by autonomic nerves react to adrenalin: an organ which shows no reaction to adrenalin has no autonomic nerve-supply. And an organ which reacts to adrenalin always reacts in the same way to nervous excitation. Also, if the organ be one much used by the animal during life, adrenalin causes marked contraction: if the organ be not much used by the animal during life, adrenalin causes little or no contraction. The action of the organ to autonomic nervous impulses and to chemical stimulation by adrenalin appears to be identical. This brings us at once to the question as to whether the chemical or the nervous impulses are the more impotent.

We know that a continuous supply of adrenalin is necessary to maintain the tone of the cardio-vascular system, and that if this be interrupted, cardio-vascular paralysis and death result. The nervous impulses to the cardio-vascular system, however, all pass through certain ganglia, the stellate and the superior and inferior mesenteric, and it has been shown by Elliot that these may all be removed in the cat and yet the animal lives. It is, in fact, of more importance to the organism for the prevention of cardio-vascular paralysis that there should be a continuous supply of adrenalin from the suprarenal glands rather than an intact nerve-supply. If this point be clearly grasped, the full significance of the changes I have already described in the suprarenal gland in diphtheritic toxæmia and other conditions will be realised in their proper significance. A continuous supply of adrenalin is necessary for life. Any cause which throws a strain upon the organism, and produces temporary exhaustion by interrupting this continuous supply of adrenalin, will result in cardio-vascular paralysis and death. The condition of the heart muscle in acute heart failure in diphtheria has been investigated by several observers. Bolton considers the acute heart failure of diphtheria is essentially a "primary failure of the heart," and is associated with fatty degeneration of the cardiac muscle and acute degeneration of the cells of the vagus. Similar changes have been described by other workers, but it is still doubtful what is their proper significance. Cowan, who has made a careful study of the cardiac changes in acute diseases generally, points out that the heart is liable to be affected in various ways, and all the elements—muscle, endothelium, connective tissue, and vessels—may be injured. Granular degeneration of the

muscle cells is almost always found, and has as its result a marked weakening of the power of the heart. Fatty degeneration is not rare, but its connection with acute disease is not necessarily close, as it is more often produced by other causes which may have been active before the onset of the fatal illness. Fatty infiltration of the cardiac muscle may be found present after death from acute disease, though of course it has no connection with it. Cowan is inclined to think that fatty degeneration of the heart is not due to the action of toxins on the heart, but to defective oxygenation of the tissues, and the toxic causes are assumed to act by their interference with this function. It has not yet been proved that the acute heart failure of diphtheria is due to the action of the toxin upon the heart muscle.

Addison's disease is one of the very few derangements of internal secretion about which we can claim any exact knowledge. It is now proved that the essential lesion is situated in the suprarenal capsules, and it is at present universally accepted that the train of symptoms characteristic of this disease is the result of the alteration in the function of these glands produced by such lesions.

This disease was first described by Dr Thomas Addison, Physician to Guy's Hospital, in 1854, while he was trying to discover the cause of pernicious anæmia. Curiously, his work failed to gain the recognition it deserved, and even when he published, six years later, his now famous monograph upon the "Constitutional and Local Effects of Disease of the Suprarenal Capsules," it failed to obtain proper notice. The general acceptance of Addison's remarkable observations was mostly due to the unselfish efforts of Sir Samuel Wilks, who collected and described a large number of further cases in 1862 and 1865. Addison, himself, considered that all lesions of the suprarenal glands would produce the same result, since this result depends, not upon the nature of the lesion but upon the interruption of some particular function which these glands produce in health. Sir Samuel Wilks, however, considered that all genuine cases of Addison's disease are due to one and only one lesion of the suprarenal glands, namely, what is now termed "tuberculous."

The discovery by Schäfer and Oliver of the blood-pressure and other actions of the extract obtained from the suprarenal glands, marked an important advance in our knowledge, and at once placed the pathology of Addison's disease upon a definite scientific basis. They also showed that the suprarenal glands obtained from a case

of Addison's disease, post mortem, did not possess this principle, the absence of which is thus responsible for the disease.

In the great majority of cases Addison's disease is the result of a tubercular infection of one, or more commonly both suprarenal glands. But it may also result from destruction of the gland by tumour formation or by inflammatory conditions or by simple atrophy. The tuberculous fibro-caseous condition is found in about 74 to 80 per cent. of the cases, and it is interesting to note that it is very frequently the only tuberculous lesion found in the body.

There are, however, a number of cases on record, which have been considered by various writers as throwing some doubt upon the constant associations of lesions of the suprarenal glands with the symptoms of Addison's disease. These may be classified as follows —

1. Cases of destruction of the suprarenal glands without any symptoms of Addison's disease; and,

2. Cases with typical symptoms of Addison's disease without any post-mortem lesion in the suprarenal glands.

In the attempt to find an explanation of the latter cases, the lesion has been thought to have been situated in the semilunar ganglia and neighbouring sympathetic plexuses, and we may thus divide these conditions into—

(*a*) Cases in which the semilunar ganglion had been found to be affected, and

(*b*) Cases in which the semilunar ganglion was normal. Let us discuss these in detail.

1. Cases in which the patient showed no symptoms of Addison's disease during life, and yet both suprarenal glands were found extensively destroyed at death, are not very numerous. It must here be recognised that the important symptom, often the first one to raise suspicion of Addison's disease, is the presence of pigmentation. Asthenia, loss of cardio-vascular tone, and diminished blood-pressure may be present and completely overlooked from this point of view if pigmentation be absent. Pigmentation requires usually some little time to develop, and if the case run an acute course it may be absent altogether or escape notice, and thus the diagnosis of Addison's disease be overlooked during life. On the other hand, if the glands be only partially destroyed, sufficient medullary tissue may remain to carry on the proper function, especially with the help of the accessory chromaffin cells.

2. Cases in which the symptoms of Addison's disease were typically present and the suprarenal glands yet found normal after

death are extremely rare. Only one or two have been fully recorded, and the glands of none of these cases were examined by the bichromate or blood-pressure methods, and thus we cannot say that although the medullary cells appeared normal they contained adrenalin.

Many observers have reported cases in which they found post-mortem changes in the suprarenal glands, but not sufficient in their opinion to be completely responsible for all the symptoms of Addison's disease, and thus there gradually grew up a school of workers who shifted the primary lesion from the suprarenal glands to the adjacent semilunar ganglia. The question of the involvement of the sympathetic system has been much disputed, and is even now not at all accurately understood. Many observers have recorded pronounced changes, and many others have been unable to detect any deviation from what they regarded as normal. Some writers have thought that both lesions are actually usually present, and each presents a definite series of symptoms.

It is an extremely difficult thing to be certain of the significance of the changes found in the semilunar ganglia in Addison's disease. I have myself made a very full investigation of this subject and studied the histology of the cells in the semilunar and other sympathetic ganglia at different ages. At a very early age these cells become loaded with pigment (as early as at birth) and surrounded by satellite cells. Their nuclei become eccentric, double, and often absent altogether, and at puberty it would almost appear, judging from a histological examination of the cells alone, they were atrophied and non-functionally active. This renders conclusions drawn from a microscopic examination of the cells of the semilunar ganglia in Addison's disease exceedingly doubtful. And as this ganglion is so liable to be also involved in the inflammatory processes surrounding the diseased suprarenal glands, secondary changes in its cells are also produced. It is of course to be expected in cases where a venous exhaustion of the gland has taken place, that the cells in the semilunar ganglia should show changes typical of exhaustion also.

Addison's disease usually is considered to run a somewhat chronic course, but several cases are on record in which the total duration of the symptoms was only a few days. A case is recorded by Lecky in 1906 in which the disease only produced marked symptoms during the last eleven days of life, but as pigmentation was present the disease was correctly diagnosed during life.

A still more acute case came under my own observation in 1910, the patient being a young woman who was admitted to the Royal Infirmary, Edinburgh, complaining of great weakness. She had been at work up till the previous day as usual, and came home with a slight headache. Next day the headache was worse, she was extraordinarily tired, and perspired profusely. Then she came to the Infirmary by car, but was unable to walk to the Medical Outpatient Department. Her previous health had always been good, and she was a basket-maker by trade. She was immediately put to bed, but the weakness rapidly increased, the blood-pressure became very low, and she died next morning. There was only very slight pigmentation. At the autopsy one suprarenal was completely destroyed by a tuberculous lesion; the other showed complete absence of adrenalin by the bichromate stain.

What is usually termed "Addison's disease" is to be regarded merely as one of the most chronic forms of suprarenal inadequacy. Suprarenal insufficiency is an extremely common condition, and is liable to occur as the result of any strain suddenly placed on the organism. Any acute fever tends to produce this condition, and as the continuous flow of adrenalin into the blood stream plays such an important part in the maintenance of the cardio-vascular tone, its cessation even for only a short time is liable to produce serious effects, and if the gland is unable to recover quickly death must take place. If the gland be greatly exhausted but still able to slowly recover its function, then the illness will be followed by a period of long convalescence, as may occur in typhoid fever, or by great weakness as after influenza.

The therapeutic problem is to replace the diminished supply of adrenalin, and thus tide the gland over this critical period until it is able to recover its function. The difficulty is that to do this successfully it is necessary to give the adrenalin continuously in small quantities. This is not easy to do. Its administration by mouth does not answer this purpose well, although it must be remembered that it is not digested by the gastric juice. Subcutaneous injection is liable to constrict the vessel locally and prevent absorption to some extent, while intravenous injection is not always practical or advisable. Possibly adrenalin given in dilute solution by the rectum may be capable of gradual and slow absorption.

Discussion

The President thought they must all agree that they had listened to a most interesting and very able paper from Dr Bruce. They much regretted that they had not had the epidiascope nor a blackboard to enhance the interest, but even without them the interest had been great. The subject was an interesting and difficult one, and on looking back some of them no doubt wished they had had before some of the knowledge which had been revealed in Dr Bruce's paper.

Dr Byrom Bramwell felt sure the Society would agree that they were extremely glad to see that Dr Ninian Bruce was so successfully following in the footsteps of his distinguished father. Much of Dr Alexander Bruce's best work was brought before this Society, and he hoped this contribution was only the beginning of a long series of valuable contributions from his son, who promised to be equally distinguished.

Personally he had been very much interested in the subject of Addison's disease during the whole time he had practised as a physician. As Dr Bruce said, Addison himself thought the disease might be due to any lesion of the suprarenal capsules, such as cancer. Wilks very strongly opposed this, and his views were followed by the Guy's Hospital staff.

In the year 1877, Dr Bramwell published a case in the *British Medical Journal* (vol. i. p. 256) in which disease of only one suprarenal was present with pigmentation ; it was a complicated case, and being a young man, he did not venture to lay too much stress upon it. That paper was criticised by the then lecturer on physiology at Guy's Hospital, now a very distinguished physician ; he stated that Addison's disease was never due to destruction of one suprarenal. On looking up the literature, Dr Bramwell found that three cases were recorded in Dr Greenhow's work on Addison's disease in which one suprarenal capsule only was diseased, and that one of these cases had actually been observed by the very gentleman who had criticised him. It must, however, be admitted that these cases were extremely rare. It was difficult to reconcile some of the post-mortem findings with the clinical features. Cases were occasionally met with in which both capsules had been completely destroyed and in which the typical symptoms of Addison's disease, such as pigmentation of the skin, were not present ; cases were occasionally met with in

which the suprarenal capsules were apparently healthy, so far as the older methods of examination at all events were concerned, and in which some at all events of the characteristic symptoms of Addison's disease were present—cases, for example, of abdominal tuberculosis involving the sympathetic. In the article on Addison's disease in his "Atlas of Clinical Medicine," he (Dr Bramwell) had referred to the observations of two French physicians who suggested that the real cause of Addison's disease was in the abdominal sympathetic and not in the suprarenal glands. In that article he suggested that some of the symptoms of Addison's disease were perhaps due to the diminished or abolished secretion of the suprarenal glands, and others to disease of the abdominal sympathetic; he still believed that that was a satisfactory explanation of the symptoms in some cases; it was perhaps supported by the close relationship which Dr Bruce had shown to exist between the suprarenals and the sympathetic system of nerves. The anomalies between the clinical and post-mortem findings could perhaps, in some cases, be explained by the presence of accessory suprarenals, the presence of which and the pathological changes in which had not been noted at the post mortem.

Dr Bramwell had likened the pigmentation of Addison's disease to the eruption of a fever. It was exceedingly difficult to diagnose a fever without the eruption, at all events before modern bacteriological developments, and it was very difficult to diagnose Addison's disease in cases in which there was no pigmentation. He had seen several cases in which there was no pigmentation or where the pigmentation was so slight it could hardly be recognised. He had always laid the greatest stress on asthenia—the general weakness, the feebleness of the heart and heart sounds, and the low blood-pressure; it was the fundamental feature of Addison's disease and was far more important than the pigmentation. It was sometimes very difficult to determine whether the skin was pigmented or not; this was illustrated by a case which came under his observation when he was an assistant physician in the Royal Infirmary. A patient came to the Out-patient Department with pronounced asthenia. His face and hands were dark, but he had been exposed to the sun. Dr Bramwell thought that the skin of his back was a little darker than it ought to be, but he could not be sure of this not having seen the patient's back before. The house-physician was asked to bring in the man's wife; they were seeing patients at the time in Harriston Lane. The house-physician went into the waiting-room

and brought in the woman ; she was asked to look at the patient's back and say whether it was darker than formerly. She said, " I couldna' say, I never saw his back before." Dr Bramwell then said, " Do you mean to tell me that you never saw your husband's bare back before ? " She replied, with some indignation, " He's no my man at all." The house-physician had brought in the wrong woman ! When the right woman was brought in, she did think that the patient's back was a little darker. Ultimately a small patch of pigmentation was detected on the buccal mucous membrane : that was a very important diagnostic point. Dr Bramwell had always attached the greatest diagnostic value to pigmentation on the mouth or tongue or under the tongue. In that case the asthenia was very marked. The patient fell down on his way to the wards ; he died two days later, and the typical lesion of Addison's disease was found in the capsules. Dr Bramwell had seen and reported some cases in which there was no tuberculous lesion : that he thought a suggestive point with regard to treatment. He had been dissatisfied with the results of suprarenal treatment in Addison's disease : the results were very different from the thyroid treatment in myxœdema. In the one of his cases in which it had produced the most marked benefit and which ultimately proved fatal, there was no tuberculous lesion of the capsules, but fatty atrophy. He had suggested that the reason for the non-success of suprarenal treatment in Addison's disease was perhaps due to the fact that in the great majority of the cases the lesion was a tuberculous destruction of the suprarenals. If the lesion of myxœdema was complete *tuberculous* destruction of the thyroid the probability was that one would not get anything like such satisfactory results from thyroid treatment as one did in that disease in which the lesion was a fibroid atrophy.

Dr Bruce mentioned a case in which he said the disease was of two and a half days' duration. Dr Bramwell thought that the symptoms had in all probability been present in some degree previously for a much longer period, seeing that the capsules were found to be caseous and completely destroyed. It was far more reasonable to suppose that in that case symptoms had been present and not observed than that Addison's disease had developed in two and a half days.

The chief point of novelty in Dr Bruce's paper was, however, the suggestion that sudden heart failure in diphtheria, influenza, and many other diseases is due to a defect in suprarenal secretion.

Dr Bramwell thought that, until further evidence was forthcoming, it must merely be regarded as a suggestion and a theory: it would be straining the facts, without further evidence, to attach any very great importance to it. The suggestion was that in diphtheria, influenza, etc., the function of the suprarenals was temporarily exhausted. This was no doubt a fascinating suggestion, but, so far as he had gathered, it was merely a theory. As regards heart failure in diphtheria, one knew that changes were frequently found in the muscle of the heart and in the nerves supplying the heart. He thought that unless these myocardial and nervous lesions were proved to be absent it would be going beyond the facts to ignore these alterations and to suggest that the heart failure was due to a defect in the suprarenal secretion. Much more information was necessary before coming to such a conclusion as that.

There were many other points which might be alluded to: one was that Dr Bruce said, if he understood him correctly, that in exophthalmic goitre there was no pigmentation. (Dr Bruce remarked that he had said one should in that case think there was a suprarenal defect.) Dr Bramwell did not think one was justified in saying that. For many years it had been well known—it was pointed out by Dr David Drummond of Newcastle—that in many cases of exophthalmic goitre there is very marked pigmentation. He had seen some of those cases examined post mortem where the suprarenals were perfectly normal.

He wished again to compliment Dr Bruce on his interesting paper: it was a valuable piece of work.

Dr Church said that Dr Bruce's paper had thrown light on many points which physicians found difficulties. No amount of reading of journals seemed to give one much information—indeed, literature was often conflicting on the actions and interactions of the hormones of the different internal secretions. Dr Bruce's paper seemed to him an epoch making one, touching, as it did, so many points of pathological interest in the cases of the ductless glands. For instance, the hormone of the secreting cells of the pancreas (hormonodrin) held in check the activity of the thyroid. Dr Church had under his care a case of exophthalmos that had been greatly benefited by the administration of hormonodrin. In the evolution of this subject he believed it would be found that one ductless gland might augment or diminish the activity of another. Possibly operation for enlarged thyroid would become more rare than it is now. Dr

Church felt that neurologists in particular were much indebted to Dr Bruce for his contribution on this difficult subject.

Professor Caird thought the Society might congratulate itself on having such a difficult subject handled so well by Dr Bruce in bringing forward his arguments. From every point of view one might regard the communication as a very able piece of work. The lucidity and logic which characterised the paper emphasises the value of experimental pathology on animals.

One would not like to say very much with regard to the paper in connection with its clinical applications, but he admitted that he was tempted, by some remarks made by Dr Bruce more than a year ago, to utilise his observations in two anxious cases. Instead of carrying out simple intravenous administration of saline, continuous subcutaneous infusion prolonged the patient's life for a greater period than usual and even raised hopes of success, which, however, was unfortunately not attained. Future attempts on similar lines may yield something better.

Dr Claude Ker said Dr Bruce's paper was important from the therapeutic point of view. If something could be got which would really make one master of heart failure in diphtheria, one would save anything between 10 and 20 lives in Edinburgh annually. When he (Dr Claude Ker) had the good fortune to hear Dr Bruce's preliminary paper, he (Dr Bruce) was not very encouraging as regards the practical treatment by adrenalin, because he then made it very clear, as he had again done to-night, that to obtain any real benefit the administration of adrenalin must be constant. That was a practical difficulty. Dr Claude Ker had gathered from Prof. Caird that he had added adrenalin to normal saline and injected it several times daily; that was nothing like a constant administration. Adrenalin had been given at short intervals of three or four hours as a drug in diphtheria for many years. It must be ten years since Rolleston introduced it, and he still held to it as a valuable drug in diphtheria. He (Dr Claude Ker) had never heard of anything to help a progressive heart failure in diphtheria. What he wanted to discover was a method of continuous administration of adrenalin. Dr Bruce had mentioned giving it high up in the rectum. It was one of the most hopeless and painful things to stand by a case of diphtheria with heart failure; the end was absolutely inevitable, and sometimes the condition lasted for several days. They had to

remember what Dr Bramwell had said, that they had myocardial lesions in heart failure—some were adrenal cases, some purely nervous cases, some muscle cases, and so on, or these might be combined. All he wished to contribute to the discussion was that adrenalin given either by the mouth or hypodermically had, in his opinion, proved a failure.

Dr Berry Hart asked if it would not be possible to try the transplantation of the suprarenal. It might be done in the lower animals first, although it did not seem a very feasible proposition in this country. That might give the desired continuous administration.

Dr Aitchison Robertson asked if it could not be given as thyroid was first given, namely, feeding the patient.

Dr Bruce replied that there did not seem to be much more for him to say, except that when one took up a subject like this and discussed the changes which take place, one was sometimes interpreted as insinuating, as it were, that this was the only structure that is diseased under these conditions: the suprarenal is only one of the glands which is affected by these conditions, the other internal secreting glands show corresponding and equally important changes. Acute processes also arise from involvement of the other internal secreting glands.

In connection with Dr Bramwell's remarks as to whether the case he had referred to could have run an Addisonian course in 2½ days, it becomes a question of what one really means by using these terms. What happened apparently was that the patient had a relative suprarenal inadequacy developing from a progressive lesion in the suprarenals and gradually destroying the chromaffin tissues. Obviously he had been getting weaker gradually, but he had a sufficient supply of adrenalin to keep him going, and he would have gone on with that until he had some strain; then the symptoms of an acute insufficiency were superposed on the chronic. The correct way would be to say a suprarenal inadequacy had existed for a fairly long time with an acute termination of 2½ days' duration.

The question of treatment by adrenalin was a very difficult one. There were a number of cases recorded in the literature in which the results had been considered satisfactory. It was quite possible that if the exhaustion was, as it were, not very great, by giving adrenalin in some form at short intervals, one might give sufficient

to tide the gland over and allow it to regain its normal function. When one says "continuous administration is necessary," that is really the ideal that one would have to aim at. It is a sufficient explanation why it is that in many cases small quantities produce no benefit whatever. Continental observers had reported cases in which small quantities have been given and proved satisfactory, and in the two cases referred to by Prof. Caird, improvement resulted. This appeared to be a method which was worth trying, but the proper way to give it continuously is a question which still requires a great deal of working out. By giving small quantities one might be able to prevent a partial exhaustion becoming complete, whereas if this were not done complete exhaustion would result, and complete exhaustion, even of quite short duration, means death.

Meeting II.—November 19, 1913

Dr John Playfair, *President, in the Chair*

This was a clinical meeting held in the Royal Infirmary.

I. Exhibition of Patients

Medical Cases

1. *Dr Dawson Turner* showed—

(*a*) Nævus of the cheek, under radium treatment, in a male, aged 17 months. The dose so far has been one of 44 milligramme-hours.

(*b*) Cheloid in a female, aged 21, under radium treatment. Patient suffered from an extensive port-wine stain, which, after treatment by carbonic snow and nitric acid, developed a cheloid in the lower portion. This extended rapidly, but has yielded to radium. The port-wine stain is also disappearing. Dose, 399 milligramme-hours.

2. *Dr Norman Walker* showed—

(*a*) A boy, aged 6, with a nævus on the flexor aspect of the left forearm. It grew gradually from a diameter of $\frac{1}{8}$ of an inch up to one inch in diameter, and then gradually began to improve, and now looks as if it had been very successfully treated by radium.

(*b*) A girl, aged 21. A fortnight before got her finger "poisoned." It was dressed, she says, with boric soap: no other antiseptic was applied. On the right elbow is a lesion like that of a burn of the second degree, a red area with a sharply outlined edge and clear blister in the centre. On the left wrist is one small, clean, round blister the size of a 3d. piece. A cast of the condition as first seen was exhibited.

3. *Dr John D. Comrie* showed—

SPASTIC PARAPLEGIA in a child, aged 7 years. Legs fairly developed, but she can only walk if supported and with legs crossed; plantar reflexes extensor. Arms strong. Visceral functions normal. Intelligence good. Wassermann reaction negative. Probably due to a congenital defect in the brain at the upper end of each Rolandic area (porencephaly).

4. *Dr Robert A. Fleming* showed—

(*a*) Allatulla, fireman, s.s. *Sarzi*. Case of BERI-BERI, wet type. History 3 weeks. Began with weakness, pains in chest, and later all over body. Leg jerks all lost. Extensive areas of anæsthesia, not confined to legs. No hyperæsthesia except slight muscle tenderness in calf muscles in particular. Dropsy of legs and feet now disappeared). Dilatation of left ventricle, but no cardiac irregularity.

(*b*) Allamulla, lascar, s.s. *Sarzi*. Case of BERI-BERI, dry type. Been ill about same time as above patient. Has much stomach pain and headache. Leg jerks all lost. Extensive and rather similar areas of anæsthesia to previous case. Rather more hyperæsthesia of muscles of calves. No dropsy. Much less evidence of any dilatation of left ventricle. Both cases feel in part on rice water. Other less definite cases amongst crew at same time.

5. *Dr Edwin Brimacell* showed—

TWO Cases of FAMILIAL NERVOUS DISEASE.

Case 1. C. G., aged 17½, a message-boy. *History*. At age of 1½ mother noticed some stiffness of left knee. Two years ago began to drag left and a little later right leg. About same time it was noticed that voice was somewhat "thick," and his hands shaky, especially on attempting to lift anything. Symptoms have become slowly though progressively worse.

Family History. 1. Male, aged 17 (patient). 2. Male, aged 14. 3. Male, aged 2 months, died of bronchitis. 4. Male, aged 11

(2nd patient);' 5, Female, aged 10; 6, Male, aged 8; 7. Male, aged 6; 8. Female, aged 4½; 9. Male, aged 2½; 10. Male, aged 1 year. No miscarriages, and no history of any similar affection or other nervous disorder in father or mother's family.

Previous History. Unimportant.

State. No signs of congenital syphilis. Mental action somewhat slow, but patient quite intelligent. Speech slow, monotonous, and markedly syllabic. Special senses, including optic discs, normal. Slight ptosis on left side, and left pupil somewhat smaller than right. A few nystagmoid movements on lateral deviation of eyes. Hands are somewhat unsteady, a definite intention tremor developing when patient lifts anything. Gait markedly spastic and a little unsteady. Knee-jerks exaggerated. Bilateral ankle clonus and Babinski sign. Abdominal reflexes absent. Lower limbs below knee, especially on right side, poorly developed, and dorsiflexors of feet distinctly weak. No lordosis, scoliosis, or foot deformity. Occasional precipitate micturition. General health good and other systems normal.

Case 2. J. G., aged 11, brother of last patient. *History.*—About a year ago it was noticed that patient walked unsteadily and was easily knocked over, that hands were somewhat shaky, so that he had difficulty in holding a cup without spilling its contents, that speech was somewhat slow, with some difficulty in commencing to articulate, and that when patient turned his head suddenly to look at anything, head shook and eyes oscillated. Walking has become more unsteady. Patient's schoolmaster advised parents to take him to see a doctor as he was unable to teach patient to write properly on account of tremor in hands. No trouble with micturition.

State. No signs of congenital syphilis. Mentally alert and memory good. Patient takes a good place at school and is not backward. Speech a little slow and somewhat syllabic. Special senses including optic discs normal. Nystagmus on fixation more pronounced in right eye. Slight irregular tremor of hands which is increased on voluntary movement. Patient walks with legs rather far apart and somewhat unsteadily. Knee-jerks brisk. Non-persistent ankle clonus and positive Babinski sign on both sides. Scapulæ slightly winged, but no weakness of muscles of shoulder girdle. No lordosis, scoliosis, or foot deformity. Other organs normal.

Dr Bramwell said that he showed these cases because of their rarity. The two patients were obviously suffering from the same

condition, the disease was thus a familial one. He regarded the cases as examples of a transition type between Friedreich's ataxia and familial spastic paralysis. Somewhat similar cases had been recorded in the literature.

6. *Dr Lovell Gulland* showed—
Four cases of ANEURISM.

Surgical Cases

1. *Mr John Fraser* showed—

TRANSPLANTATION OF FIBULA TO REPLACE AN UNDEVELOPED TIBIA in a child of 6 months. L. A., 6 months old. The lower half of the right tibia was undeveloped; the growing fibula had displaced the foot inwards at a right angle.

The fibula was divided, a portion removed to permit of straightening, and the ends wired. Into the gap of the undeveloped tibia, the fibula of the opposite leg was transplanted.

2. *Mr Lewis Beesly* showed—

Patient after left inter-scapulo-thoracic amputation for TUBERCULOUS DISEASE WITH SINUSES. *History.*—Injury to shoulder, 1905—onset of pain two months later—abscess burst, 1909. Radiogram showed tuberculous shoulder—excised by anterior method. Healed and useful arm for two years, then recurrence and fresh abscesses—amputation in Craiglockhart, 1913.

3. *Mr David Wallace* showed—

(*a*) DIASTASIS OF LOWER END OF FEMUR AFTER PLATING. W. W., a boy 12 years of age, was thrown from a horse-cake and had left leg caught and severely twisted. On admission the lower end of shaft was just under the skin on inner side, and the epiphysis was displaced forwards and outwards. Replacement under an anæsthetic failed, and therefore, after extension had also failed, plating was done on the 18th day after injury. X-rays of condition before and after treatment.

(*b*) Partial resection of stomach for CARCINOMA. W. N., a man 46 years of age, was admitted on 10th October 1913, with the history of medical treatment for 6 weeks during August and September 1913, for symptoms indicative of duodenal ulcer. He had

gained weight, but had hæmatemesis and pain after his return home. No palpable tumour. A year previously suffered from exophthalmic goitre, but no evidence of it now. 24th October, the middle section of stomach excised for a carcinoma on anterior wall, and the two portions reunited. Has made an excellent recovery.

4. *Mr D. P. D. Wilkie* showed—

Case of OSTEO-CHONDRITIS DEFORMANS JUVENILIS. Boy, æt. 8. For the past four months has been limping, the limp becoming gradually more pronounced. Has no pain, and runs about and plays football with other children. Movements of affected hip-joint free, except abduction, which is markedly restricted. X-ray shows typical appearances, viz. flattening of epiphysis and rarefaction in outer and upper part of neck of femur.

5. *Mr J. W. Dowden* showed—

(*a*) Male, aged 49. Fracture both bones of right leg, May 28th, 1913. Shortening two inches. Treated by hanging legs over the end of the bed and no splints. Marked œdema ; position and length good ; union delayed.

(*b*) Female, aged 50. Fracture of both bones of leg ; shortening one and a half inches. Treated on third day by hanging legs over end of the bed. Marked œdema ; rapid union in excellent position. No splints used.

6. *Mr George Chiene* showed—

(*a*) A woman, aged 35, married, who three years ago was treated for indigestion ; since then she has never been absolutely well, and has suffered from constipation. Several months ago she began to complain of colicy pains in the abdomen, two months ago the pain became much worse, the constipation increased, she got thinner, and did not take her food well. A fortnight prior to admission the pain became so bad that she called in her doctor ; during this fortnight she had been given suppositories, liquorice powder, cascara, large amounts of calomel (20 grains), and castor oil (3d. and 6d. bottles), also enemata every day for the last week, none of which had any result. Flatus had not been passed for a week, during which time she had bilious vomiting daily. On examination the patient is pale and emaciated, tongue furred and dry in the centre ; pulse 85 per minute, temperature 98 ; abdomen very distended and tympanitic, ladder patterns well marked at intervals, most resistance

on palpation in the region of the cæcum; rectal examination negative. Given an enema, held 1½ pints, but no result was returned. Operated on the same afternoon (11th August 1913), transverse incision below umbilicus, cæcum and large intestine greatly distended, malignant stricture in sigmoid flexure. Transverse colon brought out through incision, and colostomy performed, a Murphy's button being used in preference to Paul's tube. Gradual improvement, and sickness stopped. On the 23rd of August the patient was operated on for the removal of the tumour. The patient was put in the Trendelenberg position and an oblique incision made in the left iliac fossa; the peritoneum was divided on the outer edge, and about 8" of the sigmoid, including the tumour and corresponding mesentery and glands, were removed. An end-to-end anastomosis was then performed. A microscopic examination of the growth showed it to be a columnar-celled carcinoma. On the 9th of September the edges of the colostomy wound were freed without opening into the peritoneal cavity, and invaginated with two rows of sutures; the skin was then closed, and no leakage has since taken place. Since the operation the patient has already gained a stone in weight. The chief point of interest in the case was, that the patient's symptoms on admission were by no means severe, notwithstanding the fact that she had had absolute obstruction for a fortnight. Further, the amount of purgative medicine she had consumed in that time was enormous. Considering what she had gone through it was thought advisable at the third operation to use the simplest method of closing the colostomy wound. This has been entirely successful, as no leakage has occurred, and the bowels are moving regularly, although the patient still requires to take opening medicine.

The incisions were drawn attention to, and recommended in preference to the middle line or rectal incisions so frequently employed in similar cases.

A water-colour drawing of the specimen removed was exhibited.

B. A widow, aged 42, who was operated on in 1894 for hernia by the late Mr Annandale. In 1901 she was again operated on by him for ventral hernia; she then remained well until 1908, when a recurrence of the hernia took place. It gradually grew, but gave rise to no trouble until about three weeks before admission to hospital (September 9th, 1913), when, after some heavy lifting, she had severe pain in the right lower abdomen as well as sickness, there was no obstruction, however. She remained in bed for a week, being treated with fomentations; the vomiting ceased, and the pain

was easier, but on rising the pain gradually returned and became so severe that she came up to hospital; there was, however, no further vomiting. On admission the pulse was 80, the temperature 97; there was a large irregular swelling just above the pubis with puckered and pigmented skin over it, which bulged on coughing. In the right upper quadrant there was a hard, tender swelling, about the size of an egg. After two or three days of palliative treatment, during which time the tenderness and swelling diminished, a large transverse incision was made above the pubis, and a hernial sac, of considerable size and with numerous loculi, was opened into. The bladder was drawn up above the pubis and adherent to the hernial sac. Numerous coils of small intestine were found lightly bound together by a fibrinous exudation, which was easily severed without any bleeding occurring. The greater portion of the contents having been reduced, attention was then turned to the hard mass already mentioned; this was found to be caused by a strangulated epiploic appendix, and also a nipping of the wall of the intestine in the immediate vicinity. The cavity also contained some serous fluid, cultures of which were taken, but gave a negative result. The appendix was amputated, the excess of skin along with the sac excised, and the hernial wound closed by Mayo's method. After the patient was returned to bed a branch of the deep epigastric artery bled freely, and a portion of the skin wound had to be opened up. With this exception the convalescence was uneventful.

As far as I know, the involvement of an epiploic appendix in such a condition, and alone giving rise to acute symptoms, is a rare occurrence; I therefore thought it might be of interest to the Society to see the case.

A water-colour drawing of the strangulated appendix in the peritoneal sac was exhibited.

7. *Mr J. W. Struthers* showed—

(*a*) Male patient, aged 21 years, showing deformities due to OSTITIS FIBROSA.

(*b*) Boy, aged 9 years 7 months, after OSTEO-PLASTIC CRANIOTOMY for JACKSONIAN EPILEPSY. At the time of operation the patient was having 16 to 18 fits in 24 hours. Since operation he has been entirely free.

8. *Mr Charles W. Cathcart* showed—

(*a*) Man, after operation for tubercular glands of neck, where

internal jugular vein was torn close to the skull and closed by plugging.

(*b*) Man, finally cured after six operations for recurrent fibroid tumours of the neck and face. Subsequent carcinoma of splenic flexure of colon.

Meeting III.—December 3, 1913

Dr William Russell, *Vice-President, in the Chair*

I. Election of Members

Thomas Yule Finlay, M.D., Craiglockhart Hospital, Slateford, and Douglas James Guthrie, M.D., 8 Strathearn Road, were elected Ordinary Members of the Society.

II. Original Communications

DISCUSSION ON ISOLATION AND QUARANTINE PERIODS IN THE MORE COMMON INFECTIOUS DISEASES

Opened by Claude B. Ker, M.D., F.R.C.P.,
Medical Superintendent, Edinburgh City Hospital

It has occurred to me that the time has arrived when we may with profit investigate the rules usually laid down for the isolation and quarantine periods of the more common infectious diseases. Traditions are handed down to us, we accept them and they not only become the routine of our own practice, but are also regarded as almost sacred by the general public. And yet our views of the infectivity of certain diseases are undergoing modification, and in this subject, as in others, we are surely to be permitted to criticise the opinions of our predecessors. That is why I think that an occasional stock-taking, if I may call it so, of our regulations regarding infectious diseases might be undertaken with great advantage.

But first I wish to make it perfectly clear that, as things are at present, I totally disclaim any desire to find fault with the regulations laid down by public health departments and by schools. I have had a hand in drawing up many, and I have always felt myself tied by the views accepted by the profession at large. I do not think it reasonable to expect either a public health authority or a school

to take the initiative in relaxing generally accepted rules which appear to some of us unnecessarily strict or irksome. Speaking as a public official, if I translated into practice at once all the opinions which I have formed after long experience, I know well that not only would I cause considerable alarm and disquiet among the general public, but that I could not count on the support of the profession. It is by the conversion of the profession that progress will come, and it is for that reason that the subject should be thoroughly ventilated.

I am prepared to admit that when we go fully into the question we find that, as regards certain diseases, it is largely an academic one. An infectious case is after all confined in a room or in a hospital for two reasons—firstly, infectivity, and secondly, disability. In some conditions the infectivity, as we know, lasts very much longer than the disability, whereas in others the disability caused by the illness may be prolonged far beyond the usual period of infectivity. In the latter case the patient of course undergoes far less hardship, if the views held regarding the duration of his infection are mistaken, than he would suffer physically if he were allowed to be up and about too soon. But even here, so far as the home is concerned, a relaxation of the rules for excluding visitors and maintaining rigid isolation might conceivably be to his own advantage, as it would undoubtedly be to that of the household. In boarding-schools, particularly, much trouble might be saved. Still, in the diseases to which I allude, the time of detention in hospital is not likely to be curtailed.

Scarlet Fever.—Probably the most important and most troublesome of the infectious diseases which I propose to consider is scarlet fever. The normal minimum isolation recommended for this disease is six weeks, a period which in my own opinion may be either much too short or unnecessarily long, according to the condition of the individual case. The six weeks' tradition is not limited to this country. The most recent German book on the subject (Escherich and Schick) recommends such an isolation period, and a period of 40 days, or practically the same thing, is laid down by Wurtz and by Moizard, the two French authorities whose articles I have consulted. M'Collom, whom we may regard as a representative American authority on this subject, does not give any specified minimum, but talks of an "average" of 50 days; and we learn from Chapin that a six weeks' isolation is, if not invariable, usual in America, though that country is making advances in the direction of a shorter period.

Obviously the origin of this arbitrary regulation is the fact that in a very large number of patients desquamation may be expected to be complete in that time. The question of detention to a great extent resolves itself into this—Is desquamation infectious? If it is not, and much evidence has been collected in the last 20 years which favours such a view, the revision of our traditional ideas and regulations is overdue. If it is, it is high time that satisfactory evidence be produced to establish that fact.

Now we know that scarlet fever is infectious before desquamation begins, and we know also that it may persist for a long time after desquamation has ceased. I have never been able to comprehend, therefore, why a source of infectivity which precedes and survives desquamation should not be allowed to be responsible for infection during the desquamative period, without such infection being attributed as a matter of course to the exfoliated skin. We may justly suspect the desquamated scales in the same way as we suspect fomites, for an infectious patient may infect his skin as easily as he infects his clothes. But once he has ceased to be infectious he can no longer infect either his skin or his clothes, and when the one has been cleansed and the other disinfected I see no reason to dread a convalescent, even if he still is desquamating.

The experience of Priestley, who during an outbreak of smallpox discharged 120 desquamating children from the Leicester Fever Hospital without causing a single secondary case, did much to draw the attention of hospital workers to this subject. Millard, raising the question at a meeting of the Epidemiological Society in 1902, reported that of 21 medical officers of health and superintendents of fever hospitals whom he had consulted, no less than 10 disbelieved or doubted the infectiousness of desquamation. He himself had reduced his minimum period of isolation to 4 weeks without increasing his percentage of return cases. Shortly afterwards Hamer reported that he had gradually reduced his percentage of return cases, although entirely disregarding desquamation and adopting 4 weeks as his minimum period of detention in hospital. Moore, at Huddersfield, was able to reduce his average period of detention from 40 to 41 to 20 days without increasing his return case rate. The reports of Cameron and Turner to the Metropolitan Asylums Board also threw great doubt on the infectivity of scales at any rate late desquamation. Finally, Goodall, in his well-known book, says that "clinical evidence goes to show that after the fourth week the scales are not infectious."

The idea of the infectivity of desquamation doubtless arose from the fact that scarlet fever was considered analogous to small-pox, in which disease it is of course certain that the infective agent is present in the skin lesions and resulting crusts. But the rash and desquamation of scarlet fever are almost certainly due to the toxins of the unknown micro-organism which causes the disease and not to the germ itself. It is much better to regard scarlet fever as analogous to diphtheria, which, as has been pointed out by Pugh, Chapin, Millard, and others, presents considerable resemblances in its mode of infection and persistence of infectivity. We have the advantage here of being able to put the infectivity, as it were, under the microscope, and to determine, by obtaining negative examinations, the date at which the patient is no longer capable of causing infection. Meikle, working at the Edinburgh City Hospital, found the average duration of infection to be three weeks; Graham Smith has collected figures which point to 66 per cent. of convalescents being germ free in 5 weeks; and of course all observers have noted cases of long persistence of the bacilli in the throat. It is noteworthy, however, that in many of these prolonged cases the germs are non-virulent. Meikle emphasises the fact that in all his cases of persistence of germs the patient had large and ragged tonsils.

Now, we know that in scarlet fever, morbid conditions of the nose and throat and discharges from the ears are found, in an unduly high proportion, associated with the production of return cases. It is fairly obvious that the throat in scarlet fever is infectious from the first, and remains so in some cases for extremely prolonged periods. I have seen infection carried home by a patient detained over 20 weeks. But it does seem reasonable to assume that in a considerable proportion of cases the patient becomes, as in diphtheria, free from infectivity within 3 weeks, and that "clean" cases, with normal tonsils and no mucopurulent discharges, might be released with safety long before the statutory six weeks have elapsed. It is not, as a matter of fact, the patients who are discharged after comparatively short detentions who carry infection home. It is those who, on account of some ear or nose complication, are detained longer than the normal time in hospital who do the mischief. Mere length of detention, though not the cause of infection, will never by itself prevent return cases, which I fear we must expect in a greater or smaller percentage until we know what the causative micro-organism of scarlet fever is, and can put it, like that of diphtheria, under the microscope. In 1911 I was able to

show that the percentage of infecting cases increased steadily in proportion to the length of detention, and was most trivial in those patients who were discharged before the completion of their sixth week in hospital.

I have communicated recently with Dr Lauder and Dr Millard. The former has continued to have surprisingly good results at Southampton, employing a minimum detention of 28 days and an average stay in hospital which has varied from 30 to 32 days for the last three years. In that time 580 patients in all were treated and not a single return case occurred. I freely admit that Dr Lauder's admirable system of grading and quarantining his patients has been chiefly instrumental in procuring such brilliant results, but the most prejudiced will not deny that his figures give no support to the view that desquamation, after the 28th day at any rate, causes infection. Dr Millard does not appear to have reduced the number of his return cases, but he authorises me to say that his recent experience has not made him alter the view, which he expressed formerly, that late desquamation is harmless. His minimum detention is a day or two over 4 weeks, and his average is much the same as that of Dr Lauder. I may add that, though I have no experience of detentions of under 5 weeks, I have for several years totally disregarded late desquamation on the feet. Dr Robertson at Leith has, however, been satisfied with his experiments in the reduction of the period of isolation in hospital.

On the whole we may agree with Arnold, who, in a careful study of the question, concludes "that scarlatina is a moderately infectious disease for two weeks after the onset, but that the majority of cases cease to be infectious some time during the second fortnight, so that at the end of the fourth week only a small percentage remain so."

I would ask you then to regard scarlatinal desquamation merely as an outward and visible sign that a person has had scarlet fever. I cordially approve of the rigid isolation of such patients when they are found in a tenement or class-room in which the disease has broken out. Their subsequent detention must depend on the facts of the case. Lastly, I would suggest that even an alteration of the accepted minimum period of isolation from six weeks to five would be a great step in advance. I have some doubt whether, in view of possible complications occurring somewhat late in the illness, it would be wise to go much further at present.

As regards the period of quarantine to be imposed on contacts,

I would be perfectly satisfied with that suggested by Goodall—6 days—a period which covers the longest incubation period in my experience. Indeed in hospital outbreaks I am quite content with 5 days. Perhaps, then, for schools 7 days might be considered ample. At present it is usually longer. I cannot agree with M'Collom and those French and German writers who believe in long incubation periods in scarlet fever.

Diphtheria.—Diphtheria scarcely affords a subject for discussion as regards its isolation period. A patient must be kept until the necessary negative cultures have been obtained. Very often the illness lasts much longer than the infectivity. As regards the quarantine period, on the other hand, I have strong views. I see no use in continuing to quarantine perfectly well contacts who have given two successive negative cultures, which may well be obtained within 3 or 4 days. On the other hand I think that, as regards children at any rate, cultures should be taken from every contact and the proved carriers isolated. I have found this procedure safe and efficient in hospital outbreaks. Moreover, I see little use in quarantining contacts for a fixed period, as, unless cultures are taken, it affords no security at all.

Measles.—Our usual isolation period for measles in the City Hospital is a fortnight from admission, that is to say, from the period of eruption. Goodall gives 3 weeks from the appearance of the rash, which is, I think, unduly long. The poor condition of many of our patients, however, often makes me glad to keep them 3 weeks in hospital, if space allows. From the point of view of infectivity such detentions are undoubtedly unnecessary. My predecessor, the late Dr Wood, always allowed adults home in ten days, and in pressing cases, such as students with impending examinations, I have often followed his example. Return cases of measles, it may be mentioned, are unknown, and any feeling of restraint I may have had in my desire to discharge a patient has been due to the fear of lung and ear complications. But in hospital outbreaks I regard measles patients as free from infection as soon as the rash has disappeared, and I have never had bad results from acting up to this view. Indeed I have grave doubts if the infectivity does not cease with the catarrh, which has usually quite disappeared before the rash has begun to fade. In this opinion I have the support of Comby, who says, "We know to-day that measles ceases to be contagious after the eruption, even admitting that it is so to the end of the eruption, which is doubtful"; and of Grancher, who states,

"A case with the rash out has already become almost harmless." It is, nevertheless, amusing to find that these great French authorities recommend, the one 15 to 20 days', and the other 10 to 15 days' isolation, being, no doubt, like the rest of us, the victims of tradition. I may add that I should regard as possibly infectious after the rash cases of conjunctivitis, bronchitis, and broncho-pneumonia, although I feel that in these also the infection is probably very short lived.

I must confess that, so far as hospital detention is concerned, the question is largely academic, but in private houses a relaxation of isolation might be a great boon, even if the patient himself has still to be kept in bed. The propriety of isolation in hospital is hardly affected by these considerations, as great saving of life is annually secured by providing hospital treatment for measles. To reduce the death-rate is surely the most important function of a public health authority.

The quarantine period of measles I habitually employ in hospital outbreaks is 15 days. On one occasion only in 17 years' experience has this proved insufficient, a case occurring on the 17th day from the last exposure. In spite of this misadventure, which happened about 7 years ago, I made no change in my custom, which I notice is that recommended by Von Jurgensen and Von Pirquet. The exact incubation period of measles is extremely difficult to determine, as the moment of infection is particularly difficult to ascertain, and again, the insidious onset of the disease makes the date of sickening a matter of some doubt in a certain number of cases. For these reasons Brownlee, whose studies on incubation periods are so reliable, does not include measles among his subjects. My own experience leads me to believe that 8 days may be regarded as a minimum, and that 9 or 10 are more frequent from the probable moment of infection to the occurrence of the first symptom and from 13 to 15 days to the appearance of the rash. After all the customary estimate, 10 to 14 days, covers the ordinary run of cases. How far we might be justified in using a part of this incubation period to save school time is, of course, a matter for discussion. Elsewhere at Graz allows contacts to attend school for 8 days and keeps the infected class from the 9th to the 14th day. A similar rule is adopted by Widowitz, and something on these lines would probably work well in practice, always provided there is proper supervision.

Rubella.—Our own hospital detention of rubella is 10 days. In outbreaks of the disease in other wards, however, I have been satisfied with a much shorter time. Gärtner recommends

of 7 days from appearance of rash seems ample. Netter, with several other French authors, holds that a case is no longer infectious after the disappearance of the eruption, and says that "treatment consists in keeping the patient in his room for 2 or 3 days." Corlett is the only author whom I have consulted who seems to regard the infection as more lasting and more easily carried than that of measles. Most of the mischief is certainly done on the prodromal stage, which is often almost without symptoms.

The long incubation period of this disease would appear to justify some attempt to employ the first two-thirds of it at least at school. The limits given by Brownlee for cases collected by himself and Dr Dukes of Rugby are 12 to 20 days. This coincides sufficiently with my own experience, which does not, however, include a shorter period than 13 days. We have to remember that patients may be infectious 2 or 3 days before the rash, and this consideration complicates the question not a little, as, except for enlargement of the cervical glands, which is seldom complained of by children, the prodromal period is free from obvious symptoms. In any case I think contacts could safely attend school for 8 or 9 days after exposure, and thereafter should be excluded until the 21st day is past.

Whooping-Cough.—Edinburgh and Glasgow have for many years provided accommodation for whooping-cough as well as for measles in their fever hospitals. The terrible fatality of these diseases among the children of the poorer classes renders this a most salutary public-health measure, which has been recently imitated by the Metropolitan Asylums Board in London. No one appreciates more than I do that the long periods of time in hospital spent by whooping-cough patients are of the very greatest advantage to the children themselves, and so far from wishing to limit the practice of isolating whooping-cough in hospital, I am anxious to see it much extended. Nevertheless I have the gravest doubts as to the infectivity of whooping-cough when the paroxysmal stage has fully developed, and I consider that, in favourable circumstances and with otherwise healthy children, isolation until the whooping ceases is quite unnecessary.

Of the great infectivity of whooping-cough in its prodromal catarrhal stage there is no doubt. If a patient, who has been a week or two in one of our measles or scarlet-fever wards, suddenly commences to whoop, it is practically certain that several, perhaps many, children will be infected. But many years ago, when working

under the crowded and inconvenient conditions of the old hospital, where isolation wards did not exist, I was impressed by the fact that children admitted in the paroxysmal stage and left in very close contact with susceptible subjects did not appear to cause any infection. I have therefore taught for a long time that probably there is but little infectivity after the patient commences to whoop. This, I find, is the teaching of Henoch, who does not isolate whooping-cough when it is admitted to his children's wards, and who sees infection only exceptionally. Similar views are held by Czerny. In France, Weill, quoted by Comby, treated in his children's wards at the Charité in Lyons 20 whooping-cough cases during the year. To these were exposed 338 susceptible children, who passed through the wards in that time, and not one of these contracted the infection. All these cases were admitted in the paroxysmal stage, and Weill concluded therefore, that the prodromal stage only need be regarded as infectious. Similar experiences are reported by Guerassimovitch and by Perret and Givre, who only saw 2 cases of infection among 302 children who passed through a ward in which whooping-cough cases were mixed with the others.

If we accept the bacillus described by Bordet and Gengou as the causative organism of whooping-cough we obtain evidence which tends to corroborate this view. The bacillus is difficult to find in the sputum after the first week of the paroxysmal stage, which suggests that the sputum rapidly loses its infectivity. We must, however, remember that this bacillus has not yet been finally accepted as the cause of the disease and also that Comby, whose opinion deserves the greatest respect, states definitely that he has seen infection contracted from patients actually whooping. Peer in a recent article mentions that there is a great decrease of infectivity as the disease progresses, and that it is most infectious in the prodromal stage, and Eustace Smith expresses a similar opinion. I would be content to suggest that rigid isolation, so irksome and upsetting in a private house, is probably unnecessary after the paroxysmal stage has lasted for a week or ten days. This is not to be taken to mean that the care in looking after the patients themselves can be in any way safely relaxed.

Chicken-pox.—I have little to say on the subject of chicken-pox. I am inclined to believe that it is in some cases infectious before the eruption appears, but Brownlee appears to doubt this, and when the eruption is, as in most cases, the first symptom, we must admit that he is right. Although Comby does not regard the infectivity

of the crusts as proved, I firmly believe myself that a patient is infectious until the last has separated. On the other hand it is reasonable to suppose that when any given crust has become naturally detached, the secondary scab forming in its place is not necessarily dangerous. I often direct nurses to apply antiseptics to the raw surfaces left by crusts detached after three weeks have elapsed from the first symptom, and do not regard any subsequent scabs as infectious. Three weeks, however, appear to be a necessary minimum if this procedure is adopted.

As regards quarantine, I often allow contacts to be mixed with other children up to the 11th day, when they are isolated until the 22nd. Brownlee's studies show that very few patients develop the disease as early as the 11th or 12th days, and a still smaller number from the 22nd to the 24th. But I have always found a quarantine of 21 complete days sufficient, and Goodall is satisfied also with such a period. I therefore see no reason to change it, and I think school attendance might safely be allowed for 10 days from the *first* exposure, and be resumed after 3 weeks from the last contact with the patient.

Mumps.—The usual isolation period for mumps is 3 weeks; Comby makes it as long as from 25 to 30 days. My own experience of the disease is not sufficient to enable me to speak with any confidence, but I have never seen harm result from allowing patients out of isolation when a full week has elapsed after the disappearance of swelling. In mild cases this is often slightly within the fortnight, and as such a period would cover the time of the probable appearance of such undesirable complications as orchitis or pancreatitis, it seems that we may fairly discuss whether such a relaxation of the usually accepted rule might not with advantage be tried. In a table of Dukes, quoted by Brownlee, it is shown that of 64 cases only 1 had an incubation period as short as 13 days, and 1 of 14 days. In five, on the other hand, the period lasted from 22 to 24 days. Exclusion of contacts from the 13th to the 26th day from the date of the first and last exposure respectively would, in my view, be a quite safe rule for schools to adopt.

It does not appear necessary to consider other diseases. Bacteriological methods can be relied upon to determine the isolation period in enteric fever, and there seems no valid reason why contacts should be excluded from school. Smallpox stands by itself, every case nowadays being treated in hospital, and vaccination being imposed upon contacts. Typhus occurs too rarely to demand

consideration. Although I have come to the conclusions expressed in this paper as the result of my own practical experience, I cannot claim originality for any single idea. But I feel that some of the points raised are worthy of discussion and even if they elicit convincing proof that I am mistaken in my opinions, we will at least secure this advantage that we will know definitely that the generally accepted rules have not been uselessly imposed upon us. As it is, I fear that some of them rest upon very slender foundations. Finally, it appears to me that in these days of medical inspection of schools it should not be difficult for the early part of the incubation period of such trivial illnesses as rubella, chicken-pox, and mumps to be intelligently utilised under proper supervision.

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Discussion

The Vice-President (Dr William Russell) thanked Dr Ker for his most important communication and for his suggestions as to how the discussion should be proceeded with ; the excellence of these suggestions would appeal to every one. He believed there were present more than one head-master of the Edinburgh schools ; they welcomed them, and would be delighted to hear their opinions.

Dr Melville Dunlop said he had listened with the greatest pleasure to Dr Ker's excellent paper, and thought the Society owed him a debt of gratitude for the altogether admirable way in which he had introduced the subject. The question was one of the greatest importance, not only to the profession but to the public, and it was a matter for congratulation that it had been brought forward and opportunity allowed for discussing it from every point of view. The rules and regulations governing school attendance were so diverse and varied that it was a matter of great importance that a consensus of opinion on the subject should be arrived at. The rules which at present existed regulating these matters were very difficult to understand. Personally he often wondered whether if all the rules regulating these diseases were abolished the occurrence of fever would be materially diminished or increased ; he was sceptical on the subject, and thought there would probably be very little difference. Dr Ker a few days previously had told him that his own family, who were brought up in the closest juxtaposition to the Fever Hospital, and with whom he mingled quite freely without taking any precautions whatever, had never suffered from any infectious trouble. Sir Henry Littlejohn, too, had often told him that the district surrounding the old Fever Hospital in Infirmary Street was freer from infectious diseases than any district in the city of Edinburgh. These were very startling facts and well worth consideration.

Measles and whooping-cough were the two diseases chiefly emphasised. Dr Ker had brought the matter forward in all its aspects, and although he (Dr Melville Dunlop) thought the abolition of all rules and regulations might possibly do no harm whatever, yet he felt that these diseases were, as Dr Ker had so emphatically said, probably the most serious of all the infectious diseases which affected young children. The mortality from these two diseases was exceedingly high, and the mortality from their sequelæ was also

very serious indeed. Unfortunately the infection took place at a time when one was quite unable to recognise whether the child was going to develop one of these diseases. From a long experience he could bear out Dr Ker's statement with regard to whooping-cough, that as soon as the whoop has developed it ceases to be an infectious disease. He had had a large number of cases of whooping-cough sent into his wards suffering from pneumonia, and during the pneumonic process, as was well known, the whoop frequently ceases, only to return during the convalescence of the pneumonia; he would not like to say offhand how many cases he had dealt with, yet never, so far as he could remember, had the disease spread to any of the other patients in the ward. That, of course, is a very different thing from a child developing the whoop for the first time after coming into hospital; in that case a large proportion of children in the ward are attacked.

Then the regulations with regard to school attendance of children who have been in contact with whooping-cough and measles, he thought, required some revision. It was an extraordinary thing that, say, a little boy attending school took one or other of these diseases, an older sister, who has probably only come in contact with the boy for perhaps an hour or an hour and a half during the day, is prevented for three weeks from attending school, unless she goes to an uninfected house—what going to an uninfected house was going to do he could not say—whereas his schoolmates, who have sat cheek-by-jowl with the boy during the incubation period, are allowed to attend school. Chicken-pox, again, was such a mild disease that in 95 out of 100 cases one might ignore the rules and regulations with regard to quarantine altogether.

Scarlet fever and diphtheria were, fortunately, diseases in which the incubation was very short; they were so much dreaded by the general public that one would not, of course, like to abolish existing rules and regulations with regard to the quarantine of these conditions. Scarlet fever nowadays in nine cases out of ten was a very different disease from what it used to be; the difficulty often was to recognise that it was scarlet fever, instead of dreading it as one of the most serious infectious diseases.

How long, he asked, did Dr Ker think the infection of scarlet fever lasts? Personally he would allow these patients out at the end of four or five weeks. He supposed in the majority of cases that would more than meet the case; but, as most of those present had probably seen, a case would come back from hospital at the end

of two months, and three days later another child would be infected ; and in several cases in his experience 10 or 11 weeks had elapsed before the patient met his brother and infected him within three days of returning from hospital.

On the whole, Dr Ker had perhaps given rather a destructive criticism of these questions, and one would perhaps wish that there was a more constructive policy. It might be advisable that this Society should form a committee to consider the whole question and to formulate a set of rules which would go out as the opinion of the profession.

Dr Meikle, while considering the clinical side of the question important, said he was not going to deal with it, but would mention one or two points with regard to the prevalence of infectious diseases among school children. They caused a severe loss from both the educational and the monetary points of view, which would be lessened by a shortening of the quarantine periods. On looking over the reports of the last five years of infectious diseases in the Board Schools, he had found that the average absences due to infectious diseases, including the actual cases and contacts, equalled 4000. The schools dealt with had a population of somewhere about 40,000 ; therefore one-tenth of the school population is absent for shorter or longer periods on account of these infectious diseases. About 30 per cent. of the absences were due to notifiable infectious diseases—scarlet fever, etc.—and 70 per cent. to non-notifiable infectious diseases—measles, whooping-cough, etc. To put it in another way, one-third to one-fourth of the total absences were due to measles ; one-seventh due to whooping-cough, one-eighth to scarlet fever, and one-fortieth to diphtheria. If four weeks were taken as the average absence of these various pupils, for contacts are included, 160,000 attendances were lost, entailing a money loss of between £400 and £500 per annum, due to loss in grants. Under the new regulations a certain amount of this loss could be recovered, which, as far as local taxation was concerned, was a benefit.

In the Board Schools the " perfect attendance prize," which was a nuisance in all directions, and especially in regard to infectious diseases, had been abolished for the last three years. Then the regulations had been modified regarding the non-notifiable diseases, such as measles, whooping-cough, etc. ; they recognised that the Infant Department contained the combustible material, therefore in the upper parts of the schools the pupils who had previously had

these diseases could under certain conditions remain at school. That relaxation had not shown any disquieting results.

One difficulty was that for information regarding the non-notifiable diseases they had to depend to a large extent on reports and messages sent by parents to the schools, for in the case of the non-notifiable diseases there was often no medical attendant. This information was forwarded from the schools to the Public Health Department.

Dr Meikle agreed with Dr Ker that part of the long incubation period in many of these diseases might be used for school attendance. His difficulty was that the parents of Board School children were generally not to be relied on for accurate information about infectious disease, and if they were trusted to give the necessary information one might get into difficulty. He saw no reason why, in boarding schools especially, and also in secondary schools, part of the long incubation period of some of the infectious diseases should not be utilised for school attendance.

Dr James expressed the very great pleasure he had had in hearing the paper and his appreciation of the very great value it would be to all at the present time. He (Dr James) was in the habit of thinking that very strict regulations as regards many of these diseases had arisen at the time when the physician, instead of dealing with the matter as a clinician, began to be influenced by the *germ* of the bacteriologist. He remembered how these diseases were dealt with before the germ theory got into its present prominence. While agreeing with every word that Dr Ker said, there was one little point to which he would take exception, namely, that, as Dr Ker had stated in the billet, the "regulations for the isolation and quarantine of infectious diseases were to some extent traditional." Personally, he thought that the present-day strictness was due to the fact that tradition had been forgotten. Their grandmothers did not pay any attention to the detection of germs: the modern bacteriologist and clinician paid every attention to them. A point which might seem to tell against their grandmothers' knowledge and insight was that they considered the infective period in scarlatina was six weeks. But he contended this might be all right from their grandmothers' standpoint, but might not be so now. If the mortality statistics of scarlatina from, say, 1850 to the present time were examined, it would be found that about 1880 there was a tremendous drop in this mortality, and that this had continued.

He believed that now one was dealing with a distinctly milder form of scarlet fever from that of their grandmothers' time, so that although our grandmothers were right in considering six weeks as the infective period, it did not follow that we of the present day would be right in following their example.

Dr Meikle had referred to the action of these regulations in connection with Board School attendance. It had struck him (Dr James) that it would be of interest to the Society, and would show their appreciation of Dr Ker's paper, if some statistics in connection with the school attendance at the Merchant Company Schools were given. Dr James had (1) records from 1887 showing the number of absences of pupils suffering from infectious diseases, and (2) from 1893, records also showing the number of absences because sisters or brothers were suffering from these diseases. All the Company schools kept records, but owing to want of time he had only been able to show one, viz., that of George Watson's College for Boys. (A series of diagrams were shown, a black curved line denoting the absenteeism of pupils suffering from the various infectious diseases, and a red curved line showing the absenteeism of contacts.) Examination of these curves showed that in spite of the fact that the mortality from all infectious diseases had tremendously diminished as the result of improved conditions, yet absenteeism was seen to go on from year to year in a fairly uniform manner. This applied alike to diphtheria, chicken-pox, scarlatina, and measles, this latter causing a tremendous amount of absenteeism from year to year.

All this seemed to show that it was not the germ, but the susceptibility to it, which was at the root of these diseases. There was every year coming to school fresh virgin material for all those germs, never far away, to work upon, and the oscillations in the curves bear this out. One year a number of boys are attacked ; then there is freedom for a little ; then a new lot come to school, and there is a further attack, and so on. He believed that this would go on practically as it was doing now ; new material coming in would be constantly taking the disease. What they had to look forward to was not to stamping disease out, but to the improved conditions making it milder and milder.

With regard to the absenteeism of contacts—brothers and sisters —they had been making great improvements. In the Merchant Company Schools here was a regulation passed in the case of measles, whooping-cough, German measles, chicken-pox, and mumps, that

any pupil who was over 7 years of age, who already had had the disease, who lived in a house of three or four rooms, and who produced a certificate from a medical man to say that there was proper isolation, might continue to attend school. This was not a regulation to apply to Board Schools, but he thought it would result in great improvement as regards absenteeism in the better-class schools. Whether it would be possible to make a similar improvement as regards scarlet fever and diphtheria was another matter. If, however, they could in any way lessen the severity of the regulations as regards contacts in these diseases they would greatly improve the conditions.

Dr Burn Murdoch had been greatly interested in Dr Ker's paper, more particularly in the practical points he had brought forward. Dr Ker had asked members to give their views upon shortening the period of isolation in scarlet fever and how long one would consider it advisable for a child who had been exposed to infection to be detained before attending school. Dr Ker put it at six days. He (Dr Burn Murdoch) would like to know if he had ever seen it occur after five days. For many years past he had been absolutely happy after five clear days. When permitted he acted on this. For example, a few days ago, three girls in a boarding-school took tea in the house of a companion who developed scarlet fever the same night. The three girls were isolated for five clear days, and were then admitted to school. No case arose.

His opportunities for studying the infective period of whooping-cough had not been so great, but in the Sick Children's Hospital, when the cough had been thoroughly established and had gone on for a week or two, he could not recall a single instance where other children in the ward had become infected.

As to school attendance in measles, German measles, chickenpox, and mumps, during the period of incubation he was entirely at one with Dr Ker, and in point of fact had acted up to that for many years past. Mr Smith, the head master of Mr Gladstone's Castle School, would bear him out in this. There they were not so tied down by rules and regulations, and for long had allowed the children to attend school on the lines advocated by Dr Ker for chickenpox, German measles, and mumps. Contacts were allowed to attend during the quarantine period very much on the lines suggested by Dr Ker.

As regards the length of the infection, he had not of course had the same opportunity of observing this in measles as Dr Ker had

he agreed with him as regards the infectivity lasting throughout the duration of the rash and a short time afterwards, when no complications were present. In German measles he agreed as to the transient duration of infection.

Mr Ferard (Edinburgh Academy) spoke as one whose duty it was to administer rules of infection on behalf of a school. It would not surprise anyone present to hear that to head-masters these rules were a burden, particularly in the second term, from Christmas to Easter, when quite a material amount of time was spent in communications with regard to them. Personally he regarded their rules at the Edinburgh Academy as his marching orders, and he carried them out to the letter, in the knowledge that they had been drawn up with the best medical advice, and that it was not his business to dispute them. But for his own part he believed that many of them were of little practical use, and that they were merely an elaborate shutting of the stable door after the horse was stolen. For in too many cases the mischief was done, as they had heard that evening, in quite the early stages, before it was possible for a medical man, much less a teacher, to detect the danger. For instance, a child comes to school with a slight cold which one suspects to be the beginning of measles, but to send the child home would be to court misunderstanding with the parent. More than once a teacher had come to him and said, " I think So-and-so is going to have whooping-cough," and the child had been sent home, but at the end of two or three days he comes with a letter from the family doctor to the effect that he sees no indications of whooping-cough. The boy accordingly resumes attendance, and then a day or two after mysteriously disappears. After a few days other boys do the same, and it becomes evident that whooping-cough has once again successfully eluded the rules and firmly established itself in defiance of them.

Then, again, diseases such as chicken-pox, mumps, and German measles did not seem to him serious enough to justify the elaborate precautions taken against them, whereas, on the other hand, diseases such as diphtheria and even measles might have very serious results. In regard, therefore, to diphtheria, scarlet fever, measles, and whooping-cough, every precaution should be taken. But with regard to German measles, chicken-pox, and mumps, he continually asked himself whether it was worth while taking precautions to avoid diseases which in nine cases out of ten the children would take

anyhow. Was it not rather the duty of the school to pilot the children through them? If they did not get them during school days they would very likely do so in later life, when the result might be more serious.

A difficulty had occurred to him in connection with the suggestion that the child who had been exposed to infection should be allowed to attend school except during the period of incubation. He had before him the rules of a girls' school which had been recently drawn up. There, in the case of measles, the child might attend school up to the 8th day, but not from the 8th to the 10th after exposure; in chicken-pox not from the 10th to the 20th; in mumps not from the 14th to the 24th. In the rules of the Edinburgh Academy there was nothing analogous to that. But supposing two children, A and B, contracted mumps from a third child, C, and A developed them on the 14th day. To allow B to attend school for the first 14 days after A contracted mumps would mean that B would introduce the infection, for he would develop mumps on one of the remaining 10 days of the possible period of incubation, assuming that the original case, C, had been overlooked and A was thought to be the case through which B was exposed to infection. The only safeguard against such a contingency was not to allow B to come at all. That appeared to him to be a practical difficulty in the way of limiting absence to the actual period of incubation, and he would be glad to hear how it struck the medical men present.

With regard to scarlet fever, two things had attracted his attention in a rather serious epidemic which occurred in the Academy two years ago, when there were between 30 and 40 cases which were subsequently traced to milk from a certain dairy. The first point was that boys with sore throats were about the school for a period in some cases, as long as 40 hours before the danger was suspected, yet they did not give scarlet fever to any other boy in the school; only one other boy in the school beyond those in the two houses concerned, took the disease, and he got milk from the same dairy. The second fact was the great difficulty connected with mixed cases. Two of their most troublesome cases showed no outward symptom of any kind in the early stages. Again, a boy discharged from hospital as perfectly cured seems more properly to have carried a "return" case. In view of that second fact the question whether the period of desquamation was or was not infectious seemed comparatively unimportant, for even then was infection in cases of scarlet fever at the beginning and almost the end through the house from

nose or ear, the question whether it was infectious also in the intervening portion became a subordinate one.

In connection with Dr James' remarks, he remembered that in his first year at Edinburgh he had understood from him that in the Merchant Company Schools there were few or no rules of quarantine for infectious diseases; if this was the case it would be interesting to compare their statistics of absence through infectious illness with those of the Academy, where there were strict rules strictly administered. He believed that such a comparison would confirm his view that rules of infection too often only come into operation after the mischief had been done.

Dr Keppie Paterson wondered whether Dr Ker always felt absolutely safe in sending out his cases when there was any desquamation about the child. There seemed to be a doubt whether desquamation was infectious or not at a later stage. Would he advise the application of some antiseptic oil or ointment? On one occasion, when attending a doctor's family during an epidemic of scarlet fever which occurred several years ago, one child was sent off to the hospital. He was discharged and sent to the Convalescent Home at Musselburgh; after a fortnight there he returned home, and within three days it broke out again, spread to the rest of the family, one child almost dying. On the heels of the first patient some desquamation was seen after he had been absent from home for about eight to ten weeks, and one could not but believe that that was the source of the infection—that was the probability, at least, in those days. It was just a question whether such a result might be prevented by putting on some antiseptic application.

Dr James Carmichael wished to say a word or two in appreciation of Dr Ker's paper, the importance of which could not be overestimated. As one of the older practitioners, he could say that never in this city had any real or scientific attempt been made to learn from the experience of private and hospital practice the important lessons in regard to infectious disease. It was highly important, now that they had commenced to study these questions on the initiation of Dr Ker, who was worthy of all praise for having introduced the subject in the interests of the profession and the public, that they should take some steps, based on the valuable facts now brought forward, to give the public and the schools the benefit of their knowledge and opinions. Like Dr James he had seen the old days when

there were no school regulations at all, and the safeguards against the spread of infectious disease were entrusted to the general practitioner, and he very much doubted it in these days, with all the vexatious rules and regulations, they were very much better off than formerly. Of Board Schools he had no experience and could not therefore speak. In working-class houses isolation could not be carried out, and removal to hospital was necessary. In middle-class houses isolation under proper medical supervision could be carried out successfully. He agreed with the suggestion made by Dr Melville Dunlop that a committee should be formed to revise these rules and regulations.

Dr James Ritchie had for many years thought that the regulations for quarantine in relation to various infectious diseases should be modified. He recognised the force of Mr Ferard's statement that it was the duty of a head-master to give effect to the regulations which had been laid down for his guidance.

Firstly. With regard to periods of quarantine for those who had been exposed to infections. He had always felt safe if one who had been exposed to scarlet fever remained well after five days but he thought that the period of quarantine should be eight days, as at present. After exposure to diphtheria much school time was at present lost, because of the long period of quarantine. He agreed with Dr Ker that if two negative swabs were obtained the contact might return to school at once thereafter. As to measles he was inclined to be a little more liberal than Dr Ker because he believed that infection existed only during the catarrhal stage during the first ten days of incubation there was, in his opinion, no infection; he would, therefore, allow contacts to attend school for a week after exposure. Regarding German measles he could not speak with confidence, but he believed that the period of infectivity was of short duration. As to whooping-cough he agreed with Dr Ker that infection existed during the catarrhal stage only after that stage was past there was no infection during the paroxysmal stage. He believed that infection in chicken-pox existed only between the 12th and 21st days after exposure, and in mumps between the 15th and 20th days; that, therefore, contacts might be allowed to attend school up to the 12th and 13th days after exposure to these infections respectively.

Secondly. As to the times during which persons suffering from these diseases are infective. In relation to scarlet fever he was not

quite sure that he agreed with Dr Ker, but it was with great hesitation that he ventured to differ from him. He wished to know Dr Ker's views regarding the length of time that fomites retain power of infection. Personally, he believed that anything which had been subjected to a temperature somewhat below 212° was immediately and absolutely free from infection of scarlet fever, but if an article had not been disinfected it retained infection for a very long period. He had seen many cases which led him to believe that desquamated cuticle was infective, because he had seen fresh cases arise from contact with patients who had no ear, nose, or throat discharge, but in whom desquamation of feet or scalp had not been completed. He recalled one case, supposed to be quite free from infection; after having been home for upwards of a week the mother discovered that the scalp was scurfy. She combed it while a younger member of the family sat close by. Two days later the latter was taken ill with scarlet fever; this strongly suggested infection from the scurf. On the other hand, the speaker had recently been asked to examine a boy who had been discharged from the City Hospital some time before, with the view of permitting him to return to school. He found a large area of both feet not yet desquamated. He did not allow the boy to return to school, but three weeks later he found that five children living in family with him during several weeks of desquamation, who had not had scarlet fever, remained well. He would like to know Dr Ker's treatment of the skin; did he use any antiseptic? The speaker's practice had been to consider a patient as infective until desquamation was completed, but a case like the one just noted made him desirous of further information. Regarding diphtheria he agreed with Dr Ker. In measles he believed that there was no infection after disappearance of the rash. As to German measles he had already expressed the opinion that period of infectivity is short. Whooping-cough is only infectious during the catarrhal stage. In chicken-pox infection is in the crusts, therefore infection is present till the last crust has fallen. In mumps, so soon as the swelling had gone, infection was past; he had even seen cases in which the swelling had hardly subsided which did not seem to be infective.

Dr J. S. Fowler pointed out that the Children's Hospital and other institutions were just as much under the Public Health authority as the Fever Hospital was; they received regulations as regards the period of quarantine, etc., and had to act up to them

although they did not necessarily agree on the whole question. He did not see how any material change in the regulations dealing with infectious disease could be made until the Public Health authorities sanctioned it. With everything Dr Ker had said he thought he agreed. His only real difficulty was with regard to the incubation of scarlet fever. From his experience during the past seven years' hospital practice, he did not feel happy as regards his ward till ten days had gone without a fresh case occurring. It was, of course, extremely difficult to be sure of the exact dates of infection. He sympathised very much with what Mr Ferard said as regards the minor infections. It was no advantage merely to postpone these until the later part of childhood and youth. Mumps was almost never a serious disease in young children, the bad cases usually occurred in adolescents and young adults. It was more serious, in his opinion, for a boy of 15 or 16 to take mumps than for a baby. Again, one very seldom saw a bad case of chicken-pox in a small child, whereas many adults are attacked badly. German measles was in a somewhat different category, because of the difficulty in many cases of making a correct diagnosis.

Dr Ker, in reply, said he was surprised at the amount of support he had received. In regard to Dr Fowler's statement that he felt happier if ten days had elapsed after a scarlet fever case had occurred in his wards, it had to be remembered that there could be no safety if the source of infection was not traced and removed from the ward. If, however, that was done, in his opinion five days' quarantine was ample. He was much interested to hear that, in Dr Ritchie's experience, a mumps patient might be considered free of infection when the swelling had disappeared. If that was so, the week's interval suggested in his paper should be quite satisfactory. In reply to Dr Ritchie's remarks about return cases, he admitted the unsatisfactory state of our present knowledge, and could only hope that researches would ultimately be successful in isolating the scarlet-fever germ. He himself believed in scarlet-fever "carriers" and saw no reason to blame the skin, even if a little desquamation was present when infection occurred. Dr Keppie Paterson had asked if he treated the skin. While he did not consider it necessary, he admitted to the use of iodine which the surgeons were right should surely remove any hypothetical infection. As regards tonsils, he recalled an instance of the alleged persistence of infection in tonsils for more than 20 years which was related to him by the

late Sir Patrick Heron Watson. Nevertheless, such stories should be accepted only with great reserve, and he himself felt that it was the living human subject more than his clothes which retained infection. The really dangerous fomites were such things as cups, spoons, penholders, and throat instruments. He especially welcomed the support of Dr Burn Murdoch and Dr Melville Dunlop regarding the infectivity of whooping-cough, and was obliged to Dr James and Dr Meikle for the statistics they had given. His main object had been to feel the pulse of the profession, as it were, on this subject, and he much appreciated the support given him by the Society.

The Vice-President (Dr Wm. Russell) said that Dr Ker's communication and the discussion which had taken place were very important, and had certainly been to him an eye-opener, and when he felt in that position he always thought there must be others who felt the same. Personally he was not prepared for this important criticism with regard to the stereotyped ideas in connection with the infectious diseases, and yet member after member of the Society had got up and indicated that various of the older conceptions regarding the infectivity of these diseases were definitely wrong, and from Mr Ferard they had had a strong expression of feeling with regard to certain school regulations. Now, if that was really the expression of the views of the Society, it seemed a pity that they should separate without taking a still further step ; and he suggested that the Society should send instructions to the Council to nominate a committee, comprising Dr Ker and others, who would take up this whole question and consider the periods of infectivity of these diseases, and also the regulations at present in vogue in the various schools in Edinburgh with regard to the management of infective maladies. (*This was agreed to.*)

Meeting IV.—January 21, 1914

Mr James W. B. Hodsdon, *Vice-President, in the Chair*

I. Election of Members

William Edmund Cooke, M.B., Ch.B., D.P.H., Superintendent, Ochil Hills Sanatorium, Milnathort ; John Guy, M.D., D.P.H., Tuberculosis Officer, City of Edinburgh, Drumness, Colinton ;

Alexander Wilkie Millar, M.B., Ch.B., 6 Blenheim Place; Alex-Alexander William Neill, M.D., Craig House, Morningside Road; Dermid Maxwell Ross, M.B., Ch.B., Royal Asylum, Edinburgh, were elected Ordinary Members of the Society.

II. Demonstration of Instrument

Dr Alexander Goodall showed—

A modified trocar and cannula.

III. Exhibition of Patient

Dr Chalmers Watson showed—

Case of diabetes mellitus: to illustrate a suggestion regarding investigation and treatment.

IV. Exhibition of Specimen

Mr Scot Skirving showed—

Vesical calculus of unusually large size. About the size of a swan's egg; weight 11½ oz.; removed recently with great difficulty in India by army surgeon by supra-pubic method from bladder of native girl, æt. 10; was firmly adherent to vesical floor; good recovery.

V. Original Communications

1. FURTHER OBSERVATIONS ON PRIMARY CARCINOMA OF THE APPENDIX

(With Lantern Demonstration)

By James M. Graham, M.B., F.R.C.S.,
Assistant-Surgeon to the Deaconess Hospital

Cancer of the appendix presents several distinctive features, and in its usual form differs from the typical cancer of other parts of the intestinal tract both in its naked-eye and microscopical characters. Whilst always showing local signs of malignancy it rarely gives rise to metastases, and possesses a benignity which recalls rodent cancer of the skin. It is frequently associated with inflammation of the organ, but there are no characteristic symptoms of the disease. A further peculiar feature is the relative frequency with which young individuals are affected, and in respect of its early-age incidence cancer in this situation appears to be unique.

In a former paper [1] I recorded six cases of primary cancer of the appendix; recently I have examined five additional specimens, and as the subject is one of considerable interest I have ventured to bring my observations on these cases before the Society. Although in 1906 Rolleston and Jones [2] were able to collect only 37 cases, they expressed the opinion that with routine microscopical examination the number of cases would rapidly increase. This prophecy has proved correct, and already over 200 cases of the disease have been recorded.

As regards the frequency of the condition, I may mention that during the last two years I have detected cancer in six cases out of a series of 500 unselected appendices, all of which had been removed at operations. Cancer was therefore present in 1.2 per cent of these cases. These figures correspond fairly closely with those of other recent observers. Baldauf [3] found cancer in 1 per cent. of inflamed appendices. MacCarty and M'Grath,[4] in a series of 5000 appendices representing the material of the Mayo Clinic, reported cancer in 22 specimens, or in ·44 per cent. of the cases. As the tumour is generally small, it is easily mistaken on casual examination for a concretion embedded in the lumen, or for a diverticulum or localised inflammatory nodule. It may be so small as to escape notice altogether unless the appendix is slit up. In five of my series of eleven cases there was nothing in the external appearance of the appendix to suggest a tumour, and in one case the tumour was so minute that its nature was confirmed only on microscopical examination. In many of the cases, especially when the tumour is situated away from the tip, inflammatory changes are likely to be present which may further tend to obscure the presence of a neoplasm. As cancer rarely gives rise to symptoms or causes death except from appendicitis, it is seldom suspected. These facts explain the apparent rarity of the condition as reported by pathologists from post-mortem examinations. Out of 172 cases of primary cancer reviewed in the literature, 20 specimens were found at autopsies, and only one of my own cases was obtained post mortem. The experience of Letulle [5] suggests that these figures must represent only a proportion of the cases, as in a series of 700 post-mortem examinations where the appendix was specially investigated, cancer was detected in 7 cases (·87 per cent.).

A further reason for the extreme rarity of the condition, unless when specially looked for, is the apparently benign nature of the growth in the great majority of cases. This feature has been

commented on by most writers on the subject, and some observers have hesitated to classify the tumours as true cancers.

In certain cases the tumours are obviously cancers resembling in structure the cylindrical-celled or colloid cancers of the intestinal tract, but these cases are exceptional. Most of the tumours are of a spheroidal-celled type. Neugebauer [6] was of the opinion that these tumours were endotheliomata. In my own series of cases there is sufficient evidence to show that the cells are of epithelial origin, and their cancerous nature can be concluded from the marked degree of anaplasia and from the local invasion of the tissues. Forms which are transitional between the spheroidal-celled cancers and the cylindrical adeno-carcinomata also occur. It is difficult to ascertain accurately the relative frequency of the various types as similar histological types have sometimes been recorded under different names. From an analysis of 172 cases the relative frequency of the main types is approximately as follows:—

	Cases.	Per Cent	Average Age
Spheroidal-celled Cancer .	127	73.8	27.2
Transitional	10	5.8	20.8
Adeno-carcinoma . . .	31	18	30.5
Colloid	4	2.3	44.7

The average age of the patients ascertained in 152 cases is 30. At the same time it must be noted that cancer is not peculiar to youth as cases have been recorded at the ages of 5 and 81 and at most ages between these extremes. Reference to the above table indicates a difference in the average age incidence between the various types of cancer. A further contrast between the main types is in the degree of malignancy. Recurrence of the disease either in the cæcum or in the form of abdominal metastases is extremely rare in the cases of spheroidal-celled or transitional cancers. Spread beyond the appendix was recorded in only one case, reported by Légars [7] in a patient æt. 27, out of a series of 137 cases of these types; a few cases, where the primary origin of the cancer in the appendix was doubtful, have not been included in this analysis; but, even if doubtful cases were considered, the proportion of cancers of the spheroidal-celled group which showed signs of malignancy other than local remains extremely small. The adeno-carcinomata

and colloid cancers are less benign, for, out of 35 cases of these, types spread to cæcum or metastases in the abdominal glands or viscera occurred in 6 cases.

It is interesting to note that the age of the patients in the latter group of cases seems to be an important factor in determining the malignancy of the growth. In the 6 cases mentioned above the average age was 51.8, compared with the average age of 40 calculated from the 35 cases comprising this group. The prognosis therefore must be guarded when adeno-carcinoma or colloid cancer is found especially in middle-aged patients. These facts are well illustrated in my own series of cases. In the following table it will be seen that 10 of the cases belong to the spheroidal-celled or transitional cancers, which may be grouped together ; whilst the remaining case is a cylindrical-celled adeno-carcinoma with metastases.

Cases.	Sex.	Age.	Site.	Type.	Metastases.
1	F.	22	tip	Spheroidal-celled	No
2	F.	21	,,	Transitional	,,
3	M.	29	,,	,,	,,
4	M.	25	,,	Spheroidal-celled	,,
5	F.	16	near tip	,,	,,
6	M.	25	,,	,,	,,
7	M.	34	middle	,,	,,
8	F.	27	,,	,,	,,
9	—	—	,,	,,	,,
10	F.	19	,,	,,	,,
11	M.	48	,,	Adeno-carcinoma	Yes

A general description will first be given of the cases of spheroidal celled cancer. The case of adeno-carcinoma is exceptional and will be described later in detail.

Cases of Spheroidal-celled Cancer

The most striking facts about these cases are their benign nature ; the golden-yellow colour of the tumours, the presence of lipoids in the cancer-cells, and the intimate relation between the tumours and the lesions of appendicitis.

Sex.—Of the nine patients, where the sex was ascertained, 5 were women and 4 men. Out of 160 cases in the literature 95 were females and 65 males.

Age.—The average age in 9 cases is 24.2. It is remarkable that the patient in only one case was over the age of 30.

Site.—The tumour was situated at or near the tip of the appendix in 6 cases, in 4 cases towards the middle third of the organ. The frequency of occurrence in the various sites from a large number of cases in the literature is as follows: at or near the tip 64.3 per cent., intermediate parts 19.3 per cent., at the base in 10 per cent., diffuse throughout the appendix in 6.2 per cent.

Macroscopic Characters.—The tumours varied in size from a nodule, smaller than a split-shot, to a mass which was 2 cm. in its longest diameter. In 5 of the cases when the growth was at or near the tip it formed an obvious firm swelling; in 6 cases the tumour was not apparent till the appendix was laid open.

In 8 of the specimens the cancer completely replaced the lumen of the affected portion; in 1 case it projected into the lumen as a minute nodule, and in the remaining case it surrounded the lumen, which was narrowed but pervious.

The cut surface of the tumours was smooth and presented in 8 of the 10 cases a characteristic golden-yellow colour; in the exceptional cases the colour was greyish-white. The distribution of the growth was mainly in the position of the mucous and submucous coats, but in certain cases extension to the muscular and peritoneal coats could be seen. Inflammatory changes of a more or less marked nature were present in practically all the cases.

Microscopical Characters.—The amount of stroma varied in the tumours of this series and in different portions of the same tumour. In one case, when the tumour was grafted on the base of a chronic ulcer, the stroma was very fibrous and the cells were collected in small groups, as in a dense scirrhous cancer. In 4 cases the stroma was not so marked as in the first case and the collections of cells were larger and more numerous. In 3 cases, with the exception of a few areas of scirrhous cancer, the tumour was of a very cellular type, corresponding to an encephaloid cancer of the breast. The appearance of the 2 transitional cancers was much the same as in the previous cases, but a small area was present in each case in which the cells were either cubical or cylindrical in type, and were arranged in duct-like spaces, as in a tubular adenocarcinoma.

Except in the areas of columnar epithelium referred to, the cancer cells were small, round, polyhedral, or slightly spindle-shaped, and were closely packed in alveoli of varying size. The nucleus stained darkly and the protoplasm was scanty; mitosis was not marked.

Vacuoles were present in some of the cells, particularly of the larger cell masses.

Whilst the origin of the cancer could not be traced in any of the cases, evidence of the glandular derivation of the tumours is supplied by the columnar epithelium and duct-like spaces in the transitional cancers. The naked-eye and microscopical characters of the spheroidal-celled and transitional cancers were identical, and it was obvious that they belonged to the same group ; the deduction as to the origin of the cells of the purely spheroidal-celled cancers from the glandular epithelium seems therefore justified.

Local signs of malignancy were present in all the cases. In the earliest stages groups of cancer cells invade the submucosa and deepest portion of the muscularis. Later narrow columns of cancer cells permeate the lymphatics of the muscular coat and spread outwards to the peritoneal surface. In several cases all the coats of the appendix at the site of the tumour were invaded, and cells had reached the mesenteric fat, but no extension along the lymphatics towards the base of the appendix could be detected. In none of the cases where the lumen was obliterated could the original mucosa be traced, although a few normal tubules here and there occasionally remained. The growing edge corresponded closely with the extent of the tumour as seen with the naked eye. While the appearance is suggestive of a slow and uniform spread of the cancer there is no attempt at encapsulation.

The Presence of a Myelin Substance in the Cancer Cells.—In a former communication [1] I recorded the observation of lipoid substances in the cancer cells, particularly in the larger cell-masses and in cells which were vacuolated, and suggested that the presence of these substances, which can be demonstrated with the ordinary fat stains, explained the peculiar yellow colour of the spheroidal-celled cancers. Subsequent investigations have confirmed this view. Lipoids were absent in the 2 cases which were greyish-white in colour. In the remaining 7 cases in which the test was carried out lipoids were demonstrated in 6 cases. The absence of lipoid in the exceptional case could be explained by the solvent action of alcohol in which the specimen had been kept for a considerable time prior to the test ; in this specimen also the yellow colour had faded by the time the test was tried.

Through the kindness of Professor Lorrain Smith I have been able to examine the nature of these lipoids by means of the polarising microscope. Fresh sections of the tumours were placed upon the

heated stage and examined at different temperatures. On the dark ground of the microscope numerous minute crystals can be seen. At about 110° C. the crystals melt and on cooling down present the characteristic appearance of myelin. It may be concluded therefore that the lipoid frequently present in the spheroidal-celled group of cancers is a fluid crystal of the nature of myelin, but with an unusually high melting-point.

In the hypernephromata, which are also characterised by a golden yellow colour, a myelin substance can also be demonstrated; in both instances this is probably responsible for the colour of the tumour.

Relation of Cancer of the Appendix to Appendicitis. In these cases the cancer bears a close relation to appendicitis. Of the 9 cases operated upon 7 were diagnosed as appendicitis; one case was erroneously diagnosed as urethral stone, the symptoms disappearing after removal of the appendix. The appendix in the remaining case was removed during an operation for inflammation of the tubes and ovaries. Signs of acute or chronic appendicitis were present in all the cases.

The inflammation was acute in 4 cases, but in 2 of these there were also chronic lesions. The lesions in the remaining 6 cases were of a purely chronic nature.

In 2 of the acute cases, where the tumour was at the tip, and in one case, where it was situated in the middle portion of the appendix, the cancer had apparently grown in a previously normal appendix. The presence of a cancer may therefore influence the onset of the appendicitis, especially if, as in the last case, the tumour has grown round the lumen and caused a stenosis. The evidence in 7 cases, however, suggested that this sequence of events is usually reversed, and that cancer more frequently begins at the site of a pre-existing chronic lesion resulting from appendicitis.

In case 6 the tumour was immediately proximal to a large concretion; in case 9 the tumour was at the middle third of the appendix, at the site of a stenosis; distally the lumen was dilated and contained several concretions. In case 5 the tip of the appendix was cystic and only attached to the rest of the organ by scar tissue, immediately proximal to which was the tumour. The tumor in case 10 was minute and occupied the site of an old ulcer of the mucosa, the base of which was formed by scar tissue. In case 8 the tumour had an hour-glass shape and occupied the middle portion of the appendix; at the centre of the tumour the appendix was kinked and

stenosed. In cases 3 and 4 the tumour was at the top, and evidence of antecedent inflammatory changes were seen microscopically, dense scar tissue intervening between the tumour and the lumen.

Complete obliteration of the lumen by the cancer, whether situated at the tip or in other parts of the appendix, was a feature of all but 2 of the cases. In this respect cancer of the appendix differs from other intestinal cancers in which a trace of the lumen can always be found although much narrowed. Partial or complete obliteration of a portion of lumen is a frequent pathological change in the appendix following inflammation.

Many of the cancers commence at the site of a chronic lesion, and it seems probable that the absence of lumen, in some of these cases at any rate, is due to the fact that prior to the commencement of the cancer the lumen was altered by the process of obliteration.

This connection between obliterative appendicitis and carcinoma has been commented on by MacCarty and M'Grath, and is supported by the conditions found in the present series of cases. In 2 of the cancers at the tip dense scar tissue intervenes between the tumour and the lumen, suggesting that the cancer had begun in the remains of a mucosa in process of obliteration. In 2 cases the cancer is immediately proximal to a completely obliterated portion of the appendix. In 2 cases the appendix is constricted and pointed at the centre of the tumour where the lumen has obviously been stenosed. In a seventh case the tumour is related to a stenosis associated with a large concretion.

As there is apparently an intimate relation between obliteration and cancer, 40 specimens were examined in which there was partial or complete obliteration of the lumen, in order to ascertain whether any changes were present which might predispose to cancer. Although obliteration is apparently complete to the naked eye, in many cases the process is only partial; there may be a trace of lumen, very much contracted, often tortuous, and with the surfaces in contact, passing through new-formed fibrous tissue. Occasionally a few tubules alone remain surrounded by a fibrous and thickened submucosa. If cancer developed in such situations it would be easy to understand the obliteration of the lumen by the tumour. No constant change is observed in the epithelium in the process of obliteration. The tubules are often compressed and distorted, and the cells may be hypersecreting. Occasionally there is a simple hyperplasia, the mucosa may be represented by a single layer of columnar, cubical, or even flattened epithelium. In cases of

complete obliteration, the mucosa covering the occluded portion is frequently of the latter type.

It seems justifiable to conclude that the irritation of chronic inflammatory processes, especially in the case of tubules which are altered during involution or in the repair of denuded surfaces, may influence the onset of carcinoma in the appendix.

From the facts observed in these cases it is apparent that cancer may commence—

1. In a normal appendix;

2. More frequently at the site of a chronic inflammatory lesion, *e.g.* (*a*) in a portion of lumen which is narrowed or partially obliterated, (*b*) at the junction of the lumen with a completely obliterated portion; (*c*) in relation to a chronic ulcer.

After History of Cases.—No recurrence was noted in any of the cases whose history could be followed. Six patients reported themselves to be in good health at periods of 5½, 3½ years, 3 years, and a few months after operation. One patient died within a few days from the associated acute appendicitis. Two patients could not be traced. In one case the cancer was found at a post mortem in a patient who had died from meningitis.

These observations, although limited to a few cases and to short periods, confirm the former statement as to the benign nature of the spheroidal-celled cancers, especially as no attempt was made in any of the cases to perform a more extensive operation than the usual appendectomy.

The main facts and conclusions regarding the ten cases of this series may be summarised as follows:

1. The average age of the patients is 24.2 years.

2. The tumours are spheroidal-celled cancers, with an alveolar arrangement, and are derived from columnar epithelium.

3. The tumours are locally malignant but rarely give rise to metastases.

4. The majority of the tumours present a golden yellow colour due to a lipoid of the nature of myelin in the cancer cells.

5. The cancer may commence in a normal appendix and cause appendicitis.

6. More commonly chronic inflammatory lesions precede the cancer and predispose to it.

7. Cancer is intimately related to the process of obliteration, and this is the most frequent antecedent lesion.

Case of Primary Tubular Adeno-carcinoma of the Appendix with

Metastases.—The specimen from this case was kindly sent to me by Mr Miles for examination. As it differs in many respects from the group of cases just described and represents a much rarer form of cancer the case is recorded in detail. The growth presented none of the naked-eye characters typical of the spheroidal-celled cancers. Microscopically it appeared to be closely related to the columnar-celled cancers of the large intestine.

The patient's symptoms were specially referred to the right iliac fossa, and the incision was made there, but a correct diagnosis was not possible before operation. The fact that the patient had suffered from gastric symptoms made it important to exclude the possibility of the cancer in the appendix being secondary to a carcinoma of the stomach.

The absence of any definite tumour in the stomach made this unlikely, and the pathological changes in the appendix confirmed the view that the cancer was primary in the latter situation.

History and General Facts.—Male, æt. 48, farmer. Had always been troubled with his stomach, but more especially during the last 2¼ years. Appetite was poor, suffered from waterbrash and had pain after food. Was sick once or twice weekly. For the last month had suffered severely from pain all over the abdomen accompanied by diarrhœa. On deep pressure the patient complained of pain over the right iliac fossa, half-way between the anterior superior spine and the umbilicus; there was tenderness also over the umbilicus and at the ensiform. Some fulness could be felt in the right iliac fossa, and a small hard lump was palpable at the umbilicus.

Operation by Mr Miles on 31st January 1913. The cæcum was found to be invested by adhesions, especially in the region of the appendix. The adhesions were separated and the appendix was found to be very hard, suggesting a malignant condition; an indurated mass extended into the cæcum, and a portion of the latter was removed with the appendix. Several nodules of malignant disease were felt throughout the peritoneum, the nodular swelling at the umbilicus being one of these. A portion of adherent omentum and an enlarged gland at the base of the appendix were removed. The patient left hospital on 17th February 1913 and died one week later. There was no post-mortem examination.

Pathological Report.—The material received included the appendix adherent to a portion of cæcum, a portion of omentum, and a lymphatic gland, about the size of a bean, from the ileo-cæcal region.

Macroscopical Characters.—The appendix is 7 cm. in length.

The middle third is occupied by the tumour and is enlarged, firm, and fusiform. The peritoneal coat is intact, except towards the mesenteric side, where a mass of cancer, continuous with the tumour, has erupted and extends to the wall of the cæcum.

Distal to the tumour the appendix is atrophied, measuring .4 cm. in diameter; transverse sections show that this portion has undergone complete obliteration. On the proximal side the appendix measures .6 cm., and the lumen, although patent, is much attenuated.

The tumour has expanded the muscular and peritoneal coats and is greyish-white in colour. No trace of lumen can be seen throughout the tumour. Towards the mesenteric border the individual coats are invaded and their outline is obscured. At the centre of the tumour the diameter of the appendix is 1 cm. The continuation of the growth is of less extent and tapers off towards the cæcum.

Microscopical Characters.—Changes are present in the portion of appendix distal to the tumour, indicative of a complete obliteration of long-standing. The mucosa and submucosa are replaced by fat and dense fibrous tissue; the muscular coat is atrophied and the peritoneal coat is thickened. Immediately distal to the tumour the process appeared to be more recent, as the fibrous tissue replacing the mucosa was less dense and contained fewer fat cells. Proximal to the tumour the process of obliteration is incomplete; the lumen is narrow, and the mucosa is atrophied, the tubules being small and few in number. The submucosa contains an excess of fat, and the muscular coat is thin and fibrous.

The stroma of the cancer is composed of well formed fibrous tissue and is moderate in amount. The epithelial cells are of low columnar or cubical type. Although the cells are small they resemble both in appearance and arrangement many of the columnar-celled cancers of the colon. The appearance is that of a tubular adeno-carcinoma, the cells forming irregular gland or duct like spaces without a basement membrane. A considerable amount of mitosis could be seen. There were no lipoids as in the spheroidal-celled cancers. The main mass of the growth lay within the muscular coat, which was invaded at several parts. Opposite the attachment of the mesentery, where the cancer had erupted, the outer coats were almost completely replaced, although their outlines could still be traced. Towards the cæcum the growth rapidly diminished in size. The cæcum itself was entirely free from any trace of cancer, the mucosa was perfectly healthy.

It will be noted from the above description of the appendix that the growth was situated at the junction of a partially obliterated with a completely obliterated portion of the lumen. The main mass of the tumour expanded the middle third of the appendix, and probably its spread towards the cæcum was favoured by the presence of adhesion resulting from old appendicitis. Numerous transverse sections showed that the cancer was spreading in a longitudinal direction also, mainly towards the base but also towards the tip by permeation of the lymphatics, chiefly in the submucosa. This method of spread accounted for the fusiform shape of the growth. At a considerable distance from the centre of the tumour, near the base, isolated lymphatics packed with cancer cells could be seen. Some of the lymphatics in the mesentery were also invaded, and the gland draining them at the root of the mesentery was involved. The histological appearances in the nodules in the omentum were similar to those described in the primary tumour.

The examination of the pathological material, and especially the distribution and method of spread of the cancer, confirmed the view that in this case we have to deal with a primary cancer of the appendix with metastases, belonging to the small group of adeno-carcinomata of a cylindrical-celled or tubular type, the particulars of which are mentioned earlier in this paper.

The process of obliteration appeared to be an important factor in the ætiology of the cancer, as in many of the cases described in the spheroidal-celled group.

The malignancy of this case accentuates the benign character of the spheroidal-celled cancers. Why the majority of cancers of the appendix, especially the spheroidal-celled ones, should be so benign is difficult to explain. Early surgical intervention on account of symptoms of appendicitis will not explain the absence of recurrence in all cases, for there are many cases of appendicitis which are not operated upon, and cancer is rare post-mortem. Spread to the cæcum would be expected in the later stages, and undoubtedly occurs, but it is unlikely that this is frequent, (*a*) because cancer is rare at the base of the appendicitis; (*b*) the common type of cæcal cancer differs from the usual spheroidal-celled cancer of the appendix; (*c*) when the appendix is involved in a cancer of the cæcum it is usually easy to recognise that the cancer has spread to the appendix from without. I have examined several cases of this kind, and in every instance the lumen and mucosa of the appendix were found intact, although frequently compressed or distorted by

cancer masses in the submucous and outer coats. The spheroidal-celled tumours are small and show little mitosis in the cells; growth of the tumours is presumably slow. Since the lymphatic connections of the appendix are free, it is probable that the relatively benign character of these tumours is due to some peculiar feature of the cancer cells, as in rodent cancer of the skin.

In conclusion, I desire to express my gratitude to Professor Alexis Thomson and to Mr Miles for placing the appendices in their collections at my disposal for this investigation. I have also to thank Professor Caird, Mr Cotterill, Mr Stiles, Mr Struthers, and Dr James Miller for kindly allowing me to examine their cases.

Discussion

The Vice-President (Mr J. W. B. Hodsdon), after thanking Mr Graham for his paper, said all would agree that he was to be congratulated on having made a very valuable contribution to pathology and clinical surgery.

Professor Caird said that cancer of the appendix was even relatively rarer than the literature would indicate. For many years all the appendices he had had to deal with had been cut from end to end and carefully examined, and yet, with the exception of the preparation which had been shown on the screen, he had only come across one other case. That case was of interest, being a retro-cæcal appendix glued to the posterior surface of the cæcum and incorporated with it. It was thought right to remove the whole of the cæcum with a small part of the ileum and make an end-to-end junction. In that case the conditions were favourable for metastasis, but the patient was now in perfect health thirteen years after operation. Curiously enough, he had removed the appendix in that patient's son for acute appendicitis. In both cases the peculiar colouration of the neoplasm was very marked.

Mr Graham, in reply, said he did not think there was anything to add to what he had said, and thanked the Society for listening to his paper.

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2. MANIFESTATIONS AND TREATMENT OF INTESTINAL TOXÆMIA

(With Lantern Demonstration)

By Chalmers Watson, M.D., F.R.C.P.,
Lecturer in Clinical Medicine, Edinburgh University; Assistant-Physician, Royal Infirmary

I

In his instructive opening of the discussion on "Intestinal Toxæmia," published in the Edinburgh Medico-Chirurgical Society's *Transactions* for 1913, Professor Russell dealt at considerable length with the scientific side of the subject, time not permitting him to consider in any detail its practical aspects. He practically dismissed the clinical aspects of the subject by stating that "there is no doubt amongst us at all as to the general proposition that the retention of alimentary excreta is deleterious and injurious to the vast majority of people. You have"—he went on to say—"isolated cases where apparently this retention of alimentary excreta has no apparent injurious effect." It is interesting to compare this statement with the definition of intestinal stasis given by Sir Arbuthnot Lane in his introduction to the discussion on alimentary toxæmia held in the Academy of Medicine in 1913. Lane defines the condition as "an abnormal delay in the transmission of intestinal contents through some portion or portions of the gastro-intestinal tract, resulting in the absorption into the system of a greater quantity of toxic material than the organism is able to deal with effectively by means of the organs whose function it is to eliminate it. The toxins circulate through the tissues, and produce definite degenerative changes in them. Finally, the organs whose function it is to convey, convert, and eliminate the several toxic products circulating in the blood themselves undergo degeneration, under the influence of prolonged and progressive strain. Then the degenerative changes in the tissues progress more rapidly in proportion to the failure of the organs of elimination."

From the general nature of Professor Russell's statement it is difficult to say what his opinion exactly is as to the *rôle* of intestinal toxæmia in the production of chronic disease. It is probable that most clinicians will agree with the general propositions stated by him, but it appears to me that on the all-important points of

detail there may be a good deal of difference of opinion. There may be some who attach as much importance to intestinal toxæmia as I do, others who think my views fanciful and exaggerated, and yet others whose ideas on the subject are less determined: and accordingly it may be instructive if we come into closer grips with the subject in its practical aspects, and to this end I propose to set forth an epitome of my views in this and subsequent articles.

In the present paper attention will be largely directed to a summary of the essential factors in the clinical pathology of the condition and the means by which the condition is recognised clinically.

The second paper will deal with clinical illustrations taken from cases which have been under my observation and treatment for lengthy periods, with a statement of the results of treatment. As at the present time my facilities for the adequate investigation of these cases along modern lines are greater in private than in hospital, I will draw, in the following records, exclusively from my private case-book.

In the third and concluding paper the question of treatment will be fully discussed, the details of medical treatment being carefully considered, with remarks on the operative treatment of cases which have proved not amenable to medical treatment.

At this date it is hardly necessary to elaborate the importance of the subject. We all know that in the past few years there have been a very large number of abdominal operations performed for chronic disease, more especially the removal of the appendix and to a lesser extent operations on the stomach in which the results of the operation have proved disappointing to the patient. It is too commonly assumed that the responsibility for this erroneous treatment is entirely surgical, in fairness the responsibility has to be shared by the physician and surgeon alike. In fact I would rather say, in connection with the class of case that I am considering, that the physician is more to blame than the surgeon, as too often the surgeon is called in, in these chronic cases, to find that the condition, whereas it is really the duty of the physician to exhaust the possibilities of modern methods of investigation and to form a provisional opinion as to the nature of the condition and the best method of treatment by education by surgical means. This subject will be further considered later.

1. *The Pathological Basis of Intestinal Toxæmia.*—Stated shortly, this consists in the following, in various combinations:—

(*a*) The presence of constipation. The existence of this is determined to some extent by the history of the patient, and also by the use of the enema and the X-rays. The X-rays have special value, in so far as they are the only means at our disposal for locat ing the part of bowel primarily affected. In this connection it is important to bear in mind the comparative frequency with which we meet in practice cases of severe constipation in patients whose bowels move, though very imperfectly, every day.

(*b*) An associated catarrh of the large bowel, and, in aggravated cases, the lower end of the small intestine. The existence of this is shown by excessive putrefaction in the stools, with frequently marked excess of mucus and, it may be, attacks of diarrhœa alternating with constipation. The urine is also frequently abnormal, containing catarrhal cells, white or red blood corpuscles, oxalate of lime crystals and bacteria, this condition of the urine being directly associated with and dependent upon the diseased state of the bowel. Particular attention may here be directed to the value of irrigation following the enema ; it is my practice in these cases to clear out the bowel with a soap and water enema, and to follow this with an irrigation of two pints or more of plain water. Under normal conditions the irrigating fluid is returned practically unaltered, or at most with a trifling amount of mucus, or mere traces of fæcal matter. In septic states of the bowel associated with intestinal toxæmia the irrigating fluid is dark, feculent, and offensive, often with excess of mucus and abnormally loaded with bacteria.

(*c*) Dilatation of the bowel, more especially the cæcum and colon, often associated with visceroptosis. These conditions are revealed by the presence of splashing on manual examination, more especially in the epigastric and cæcal regions, and by the results of X-ray examination. In cases associated with marked ptosis the contour of the abdomen in the erect posture is distinctive, being flat in its upper half and prominent and bulging in its lower part. It is probable that in some of these cases a congenital weakness of the abdominal musculature is an important factor, this weakness being aggravated by defective posture and the use of faulty clothing in the form of corsets. In cases associated with marked ptosis the stasis is aggravated by the ptosis and conversely.

(*d*) The presence of adhesions, membranes, pathological kinks, etc., mechanically interfering with normal intestinal movement, with resulting stasis in the large and small bowel. These conditions are suggested or revealed by the presence of isolated areas

of tenderness on pressure, often erroneously attributed to the appendix, and by X-ray examination.

It is not my intention to discuss the various theories or explanations for the presence of these adhesions. We are here chiefly concerned with ascertaining whether their presence is an important factor in contributing to the development of intestinal stasis and toxæmia.

The brilliant experiments of Carrel may be cited as bearing directly on toxæmia. Carrel found, from his experiments on living tissues growing in a sterile medium, that the vitality of the tissues was diminished and that the growth of the cells was slowed when the product of the cells' activity was incompletely removed. By washing away the toxic products he was able to carry their growth along a satisfactory line. In other words, by attending carefully to the drainage and by giving suitable foods, he gave the tissues a continued life and satisfactory rate of growth. The conditions that occur in the human being as a result of intestinal toxæmia are, for practical purposes, identical with the above. As a result of imperfect removal of the waste products in the bowel slowly acting poisons are absorbed into the system, and induce the clinical manifestations of toxæmia later referred to.

2. *Factors regulating Normal Intestinal Functions.* There is, I think little doubt that some people start life handicapped in the state of development and tonicity of the intrinsic and extrinsic abdominal muscles, and in such cases a lesser degree of derangement along one or other line will induce a greater degree of intestinal intoxication than would otherwise obtain. It is probable that artificial methods of dressing, and more especially the abuse of corsets, is also a contributory factor in this direction.

Another point that occurs to me in connection with this is the possible relationship of internal secretions to intestinal movement. I have long held that an internal secretion plays a more important part in the intestinal movement than is at present supposed: this opinion is based largely on the fact of the marked frequency with which we meet with cases of pre-menstrual constipation in women, and in the early occurrence of constipation in pregnancy before the mechanical cause of origin can come into operation. Diet undoubtedly plays an important part in regulating normal intestinal functions. Articles of food, such as oatmeal fruits and vegetables, rich in cellulose, are very helpful in promoting a physiological action of the bowel. At the same time it must, I think,

be admitted that in most advanced cases of constipation diet can effect little in the way of improvement.

The acquirement of proper habits in childhood with regard to the movement of the bowels and the maintenance of these habits throughout life are also important factors in maintaining a healthy state of the bowel and tissues generally. So also is the maintenance of a high level of efficiency of the internal glands, such as the liver and the thyroid gland, which are intimately concerned in neutralising the normal intestinal toxins. A normal activity of the kidneys and skin in regard to their work of elimination is also an important point.

3. *The Clinical Association of Normal Intestinal Functions.*—These may be considered under the following headings :—

(*a*) *Subjectively.*—The absence of any symptoms of ill health, digestion being performed in the painless, unconscious manner characteristic of good health.

(*b*) *Objectively.*—Attention is to be directed to the following three points :—(1) The passage of normal stools ; (2) the elimination of a normal urine ; (3) the presence of intestinal stasis.

(1) *Normal Stools.*—A normal stool consists largely of bacteria ; it is acid, neutral, or faintly alkaline in reaction, and is free of offensive odour. Under the influence of an aperient a healthy stool acquires a more distinctly fæcal odour ; in cases of intestinal toxæmia the stools may be markedly alkaline in reaction, and they are exceedingly offensive, often containing marked excess of mucus, the condition indicating abnormal putrefaction in the large bowel. When a healthy bowel is cleared out by an enema, and subsequently irrigated with plain water, the returned fluid should be practically clear ; in cases of severe intestinal toxæmia a very different result is obtained, the returned fluid being fæcal and foul-smelling, and containing great excess of bacteria. The correction of this state of the stools is usually accompanied by disappearance of the symptoms.

(2) *The Elimination of a Normal Urine.*—In this connection attention should be particularly directed to the following points, the significance of which is, I think, not sufficiently appreciated, the different condition referred to occurring in various combinations : (*a*) A deficiency in the total amount of urine passed. An extreme example of this may be cited in the case of a stout lady affected with chronic rheumatism who was under my care some years ago, the total amount of urine voided in three days being just

under twenty ounces. (*b*) The presence of cellular elements in the urine in marked excess of the normal. The examination of a centrifuge deposit of a normal urine, obtained fresh, may show the presence of two or three epithelial cells, and very occasionally a white blood corpuscle. In the cases under consideration the deposit in the centrifuge tube may be considerable, consisting of epithelial cells, white blood corpuscles, and a few red cells. (*c*) The presence of numerous oxalate of lime crystals is a feature in not a few cases. (*d*) The presence of bacteria in the urine, these

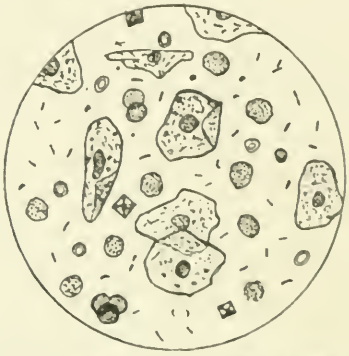

FIG. 1.—Drawing illustrating Catarrhal Cells, Pus Cells, Red Blood Corpuscles, Oxalate of Lime Crystals, and Bacteria.

bacteria being either voided in the urine or multiplying at an abnormal rate when voided, the latter condition indicating an unduly toxic condition of the urinary excretion. Fig. 1. illustrates in a slightly exaggerated form the conditions frequently met with in these cases. I lay comparatively little stress on the presence or absence of indican, the one test on which reliance is laid in text-books. The examination of the urine from these points of view requires, I think, much more attention than is commonly given to it. In connection with the bacteriological examination of the urine in men, I never feel justified in passing the catheter, as this, in my opinion, is an unnecessary proceeding, and not without danger. I rely on obtaining a sample of urine taken direct into a sterile tube from the middle of the stream, with all due antiseptic precautions.

4. *X-Rays—The Presence of Intestinal Stasis.*—In recent years considerable light has been thrown on many gastro-intestinal disorders by the application of the X-rays after a bismuth meal. Since Cannon first investigated the normal intestinal movement in animals by bismuth meals a vast amount of work has been carried out on the intestinal movements in man, notably by Hertz and others, and there is now an extensive literature on the subject. A great deal of very valuable information has been obtained, which is of value to the physician and surgeon alike, in the diagnosis of various gastro-intestinal disorders, and it may safely be said that our knowledge of the great value of X-rays in the diagnosis of abdominal conditions is still in its infancy. At the present time there are difficulties in regard to the completeness of the information gained by their use. These arise in the interpretation of the results, and are due largely to the absence of any standard method in connection with the routine X-ray examination. A further difficulty is the absence of sufficient data as to the range of normal limits in people who may be perfectly healthy. It would be a great advantage if radiographers adopted a standard bismuth meal, and if the routine examinations were made at uniformly recognised periods, as by this means we should be better able to compare the results of different observers. It is interesting to note that a committee of radiographic experts has recently been appointed by the electro-therapeutic section of the Academy of Medicine to deal with this question.

We will first refer briefly to certain elementary facts, which are generally accepted as established, in connection with the passage of bismuth meals.

Hertz, with a view to determining the rate of movement in the small and large bowel, examined eighteen normal subjects after a bismuth meal at different intervals, in two cases every hour, in the majority of cases four times a day, and once or oftener on the day following. He found that the shadow appeared in the cæcum most frequently four hours after the meal, the hepatic flexure was reached from four to eight hours, the splenic flexure at seven to fourteen hours, and the junction of the descending colon with the iliac colon from eight to sixteen hours. In the pelvic colon the rate of progress was more slow. These figures represent an average. A diagrammatic representation of these findings is shown in figure reproduced from Hertz's book on *Constipation.* My own experience leads me to regard these figures

as not reliable as a general average, and certainly not to be taken as a guide for the presence or absence of a pathological stasis.

It may be useful to follow the course of a bismuth meal as observed with the screen and radiograms. My cases have been examined by Dr Hope Fowler, the patients being examined immediately after the taking of the meal, and on four, five, or six occasions subsequently, at intervals ranging from three to seventy-two hours. Screen examinations were made in every instance by Dr Fowler and myself, a very large number of photographs being taken at the same time. The meal consisted in an emulsion of 10 ounces, con-

FIG. 2.—The Normal Rate of Movement of a Bismuth Meal. (The figures 4 to 32 represent hours.)

taining 60 grammes of oxychloride of bismuth, followed by 30 grammes of the same salt with 4 grammes of lactose in 3ii of water. Examinations were made chiefly in the vertical position.

The *shape* and the *position* of the stomach is first determined: normally the stomach hangs vertically to the left side of the middle line, and is shaped like the letter J, the lower end of the stomach reaching from a little above to a little below the umbilicus. The *tone* of the stomach is then observed; this is revealed by the manner in which the organ grips its contents, and by the rate and degree of *peristalsis*, which must be carefully noted. Immediately after food enters the stomach the organ at once commences to discharge its contents into the duodenum. The food passes into the first part of the duodenum, forming a characteristic duodenal cap, which is usually but not invariably revealed. After its passage

into the second part of the duodenum the food may be seen passing quickly through the small intestine, but its rate of movement is usually such as to preclude a photograph being taken. In favourable cases a delicate stippling of a photographic plate may be seen, indicating the bismuth contents in the small intestine. The food is next visible in bulk when it reaches the terminal coils of the ileum and cæcum, which should be reached in from 4 to 6 hours after the meal is taken. After the sixth hour practically all the bismuth meal should be in the cæcum. If a cup of tea and bread and butter be now taken it will be observed that the introduction of food into the stomach induces peristaltic movements in the lowest coils of the ileum, with a relaxation of the ileo-cæcal valve, which is followed by the discharge of the fluid contents of the ileum into the cæcum. On the following morning the bismuth contents are largely expelled ; a screen examination may reveal a shadow in the rectum, pelvic colon, or iliac colon, this representing the remaining part of the bismuth meal. Our knowledge of the mechanism of the movement of the large intestine is still incomplete. The evidence available justifies the view that intestinal movements of the large bowel take place 4 or 5 times daily, as a result of which the more solid contents in the large bowel are propelled onwards over a large area.

It may be safely assumed that in cases in which the bismuth meal is not completely voided within forty-eight hours a pathological stasis is present, the degree and situation of this stasis being determined by the situation and amount of the bismuth contents present. Thus the presence of a residue in the rectum and pelvic colon at this time may indicate a defect in the power of the rectum to expel its contents, but this is naturally a much less severe stasis than would be indicated by the presence of the whole meal, or the major part of it, in the cæcum and ascending colon at the same period. A simple means of testing the rate of intestinal movement is afforded by the use of plain bismuth or charcoal, and observing the time when the bismuth appears in the stools and the length of time that elapses before the stools resume their normal colour. This test is of value as showing the existence of intestinal stasis, but gives no information as to the site. It gives no indication as to whether the delay occurs in the terminal portion of the ileum, the cæcum, ascending colon, the transverse colon, sigmoid flexure, or the descending colon or rectum, any one of which may be the primary site of the stasis. The X-rays are the only accurate means we have of determining precisely this point.

The three most common sites of stasis are the lowest part of the ileum, the cæcum and ascending colon, and the rectum. At the present time a good deal of difference of opinion exists as to the degree of ileal stasis that is met with and as to the existence of an associated and dependent duodenal stasis. Dr Jordan, who has directed special attention to these points, attaches great importance to ileal stasis as a common pathological condition and claims that it is associated with marked slowing of movement in the duodenum, due to dragging of the membraneous attachments at the duodeno-jejunal junction. His views are based on a large series of cases radiographed by himself and subsequently observed at operations performed by Sir Arbuthnot Lane.

Hertz, on the other hand, regards ileal stasis as largely a physiological condition, and he has, further, been unable to confirm Jordan's X-ray findings of a dilated duodenum. A very crucial point is here involved, and it is highly important that it should be determined which view is the correct one. For my own part, while originally sharing the views advocated by Hertz, I have lately seen reason to change, and am satisfied, from a more adequate first-hand knowledge of the technique adopted by Jordan, that his teaching on these important points is substantially correct, and Dr Hope Fowler concurs in this opinion. Further reference to this important point will be made later in the course of reference to individual cases.

5. *Clinical Conditions induced by Intestinal Toxæmia.*—Long-continued observation of many cases of chronic disease, and more especially private cases which I have had the opportunity of investigating by the aid of modern methods of diagnosis, has led me to recognise three distinct groups of conditions that arise mainly or entirely from intestinal toxæmia. These are as follows: (1) A Neurasthenic Group. This includes some cases at present regarded as neurotic, and also a sub-group in which the symptoms are mainly mental. (2) Rheumatoid Arthritis. (3) A "Dyspeptic" Group.

The division between these groups is somewhat arbitrary, but their separate consideration is justified on practical grounds. A few words may be said by way of elaborating these points.

1. *Neurasthenia.*—In the discussion above alluded to Professor Russell, in dealing with the relationship of neurasthenia to intestinal toxæmia, gave expression to his views as follows: "In the neurasthenic we all know the marked prevalence of constipation,

so marked that I am not sure that we may not take up the position that we seldom see a definite case of neurasthenia without its being accompanied by definite and obstinate constipation. By relieving this systemic constriction and allowing the blood to get freely to the periphery you relieve the symptoms." This statement illustrates the view which is held by some writers that constipation and other gastric symptoms met with in neurasthenia are a result and not a cause of the neurasthenic condition. A different view is now held by many clinicians. This is well brought out in the course of a discussion on the application of surgery for the relief of neurasthenic conditions published in the *Transactions* of the American Surgical Association for 1910. In introducing the discussion Blake writes as follows: "While the neurologist is apt to consider the lowered blood-pressure, the disturbance of intestinal functions, the concomitant putrefaction, gas formation, and auto-intoxication as symptoms of neurasthenia, there can be no doubt but that they are in a large measure causative." I fully share the view so well expressed by Blake. In the course of the last few years I have had under my care many cases of neurasthenia, some of whom have suffered from the condition for years, in which the critical analysis of the symptoms and the careful examination of the stools, the urine, and of the digestive tract by means of the X-rays pointed clearly to an unhealthy state of the *prima via* as the primary causative factor of the disease. The cases referred to made a complete and satisfactory recovery coincident with the use of measures directed to restore the gastro-intestinal tract to a healthy condition. In some of these cases the patients had previously been regarded as neurotic, and treatment based on that diagnosis had failed to relieve the symptoms. I believe that not a few patients who are at the present time regarded as poor "neurotic" creatures are suffering from intestinal toxæmia, and the "neurotic" manifestations would largely disappear if a correct diagnosis were made and appropriate treatment applied. I have also seen in the course of the last year or two a number of advanced cases of neurasthenia associated with more or less visceroptosis which were also largely the result of a severe intestinal toxæmia. In some of these cases, which will be illustrated later, the results of medical treatment in my hands have been much less satisfactory, and in regard to them the question of the advisability of operative treatment has to be considered.

A sub-group of these cases comprises patients whose symptoms

are mainly mental. Three well-marked cases of melancholia have lately been under my care, which were clearly shown to be the results of an undetected septic condition of the large bowel. These made a satisfactory recovery when the condition was recognised and treated appropriately. In two of these cases the primary source of infection was to be found in the septic state of the teeth and gums. It is probable that most alienists could cite similar cases.

2. *Rheumatoid Arthritis.*—The late clinical manifestations of rheumatoid arthritis are too well known to need description. A visit to the medical wards of any general hospital or to a hospital for incurables at the present time gives eloquent testimony to the ravages of the disease and to our apparent comparative helplessness in its treatment. We have as yet no exact knowledge of its etiology. The clinical features of the disease and its morbid anatomy strongly suggest an infective origin. This view is now very widely held, but it must be confessed that too little attention is still directed to the adequate investigation as to the primary source of the infection in the early stages of the disease at the stage when much can be looked for from appropriate treatment.

It is of interest to refer in this connection to the comprehensive investigation into the etiology which has been going on for the past few years in a special hospital at Cambridge. Much valuable information will be found in the bulletins published by that committee in the last few years.

It is now many years since Dr Catherine Clark, at my suggestion, made a careful investigation into the visceral changes present in cases of advanced rheumatoid arthritis dying in the parochial hospital. Her investigations showed widespread changes in the kidneys, pancreas, vascular system, and tissues generally, thus proving the widespread nature of the pathological process and strongly suggesting the correctness of the view which I had long held, that the joint changes are not the essence of the disease, but are merely to be regarded as one of its most distinctive characteristics.

As a result of a close study of the disease for many years I have been led to share the view as to its infective origin, the source of the infection being invariably one of the three mucous surfaces of the body. In the great majority of cases the infection comes from the gastro-intestinal tract, oral sepsis and pyorrhœa alveolaris being not infrequently primary factors in this intestinal infection.

In the remaining cases the infection comes from the genito-urinary tract or the respiratory mucous membrane. With regard to the actual infective agent we have as yet no precise knowledge. For my own part I am doubtful whether the clinical pictures of rheumatoid arthritis, as we see them in their varied manifestations, are due to a single infecting agent. I think it probable that a slow infection by different organisms brings out the pathological conditions that are at present included in the term rheumatoid arthritis.

I am satisfied on this point, that the disease is essentially curable in its early stages, and that if early cases were adequately investigated, and the appropriate treatment applied to the main source of infection, we would soon see a profound change in the incidence of the disease in the severe forms at present met with. In the later stages of the disease the intestinal mucous membrane, and especially the large bowel, has become seriously involved, and in some cases it is not possible to correct this by medical means. Various surgical procedures have in recent years been advocated in order to eliminate the large bowel as the main source of infection. The indications for surgical treatment will be dealt with in later articles.

I wish here to emphasise the highly important part played by the gastro-intestinal tract as a source of infection in rheumatoid arthritis, and will elaborate this by detailed descriptions of cases that have been under my observation.

3. *A "Dyspeptic" Group.*—This group, arbitrarily called a "dyspeptic" group, is characterised mainly by symptoms of indigestion. Many cases of this group simulate very closely chronic appendicitis, duodenal ulcer, and gall-stones. A large proportion of patients who have in the past few years had an innocent appendix removed without relief of symptoms have undoubtedly been cases of intestinal toxæmia, in which the primary mischief was in the right lower quadrant of the abdomen, chiefly the cæcum and terminal coils of the ileum. It is interesting to refer to the change in opinion that has occurred in the past few years in regard to various gastro-duodenal conditions requiring surgical treatment. A few years ago ulcers in the duodenum were regarded largely as primary affections of the part. At the present time Lane, Mayo, Moynihan, and others agree in regarding these as mainly secondary to disease either in the terminal portion of the ileum, cæcum, or appendix, often associated with adhesions and kinks in the bowel. In Lane's view the primary trouble is a stasis in the cæcum and ileum, the

appendix trouble when present being secondary. Moynihan and others attach more attention to the appendix as the primary factor.

The change in surgical opinion which has taken place in recent years on this subject is well brought out in a quotation from a recent paper on the subject of "Intestinal Angulations and Kinks associated with Stasis," by Mr Wheeler. He writes as follows: "It is not so long since we were taught that intestinal kinks and adhesions other than inflammatory were a product of the imagination, and now those who were first to discount the possibility of the presence of kinks and adhesions have taken refuge in the statement that every individual has adhesions and kinks and they do no harm. These two contentions are impossible to adjust or harmonise. It may be now taken as an axiom that in a large proportion of abdominal cases, cases of gastric or duodenal ulcer, appendicitis, gall-stones, and allied conditions, including cystic ovary, there is present a varying degree of intestinal stasis associated with the presence of certain pathological angulations and kinks. In a certain proportion of the cases these conditions, *per se*, give rise to no symptoms whatever, in others the symptoms are slight or masked, in the remainder they are so marked that a novice could not be deceived as to their cause."

It is beyond the scope of the present paper to discuss the various theories of origin of the different membranes and adhesions, we are here only concerned with their relationship to diseased stasis. Short reference is, however, necessary to the subject.

The more important kinks that require consideration are Lane's kink near the ileo-cæcal junction, Jackson's membrane, investing the cæcum and ascending colon, the kink at the duodenojejunal junction, causing dilatation of the duodenum, and the terminal mesocolic kink in the pelvic colon. A few words are necessary with regard to these.

Lane's Terminal Ileo-cæcal Kink was described as caused by a band first forming part of the under surface of the mesentery and afterwards a distinct ligament which attached itself progressively to the ileum more and more distant from the mesenteric border. The band is shorter than the mesentery, thus causing a pucker or kink of the ileum, which is in addition often dilated on its long axis owing to the attachment of the band to the anti-mesenteric border. More than one of these kinks may be present in this region. Fig. 3, reproduced from Mr Wheeler's article, gives an illustration of Lane's terminal ileal kink.

According to Lane this and other intestinal kinks are due to "the crystallisation of lines of force, to prevent prolapse of the viscera in cases of chronic constipation."

Jackson's membrane has attracted considerable attention in the past two years. It is a common lesion found in patients suffering from right-sided pain and discomfort, with a loose, splashing cæcum, constipation, and some visceroptosis. It extends from the abdominal wall to the cæcum and the ascending colon. At the hepatic flexure the membrane when lifted up on the finger often forms a formidable band, binding and obstructing the colon (see Fig. 4). Opinions differ as to whether Jackson's membrane is a developmental structure (Reid) or an acquired pathological condition. There is little doubt, however, that it is frequently a

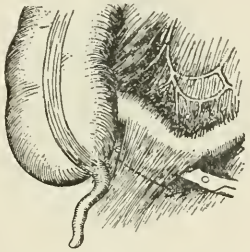

Fig. 3.—Terminal Ileal Kink (Lane).

contributory factor in the clinical pictures of stasis and associated toxæmia.

A Dilated Duodenum.—Lane has pointed out that in cases associated with marked ileal stasis the duodenum is frequently dilated. This condition is produced not in any direct manner from an ileal kink but by the formation of bands at the duodeno-jejunal flexure, or by a band running across the duodenum just to the right of the superior mesenteric artery. The frequency with which this dilated duodenum is found in cases of ileal stasis is not yet fully determined, and some writers are doubtful of its existence. Since my attention has been closely directed to this point, using the technique advocated by Jordan, I have seen the association in a marked degree on three occasions; in one of these which has since been operated on, the X-rays diagnosis was fully confirmed at the operation.

A kink of the sigmoid flexure is occasionally present, and is probably due to the shortening of the phrenocolic ligament.

A lower sigmoid kink is very common. This may not interfere with the bowel at all, but in some cases it is a definite cause of severe constipation, abdominal pain and discomfort, dilatation of the colon above, and all the manifestations of intestinal toxæmia.

A very good description of the manner of development and incidence of the symptoms in this dyspeptic group may be taken from a recently published surgical paper by Carson. In describing the development of the adhesions and their results he writes as follows: "It is characterised by the formation of adhesions

FIG. 1.—Jackson's Membrane

between the colon and the peritoneum lining the outer walls of the abdomen, which tend to cripple the peristaltic movements of the large intestine and to give rise to pain and delayed passage of the intestinal contents. The condition is associated with enteroptosis of greater or less degree, and in later stages by the formation of an adhesion, binding the lower end of the ileum firmly to the right wall of the pelvis. It is probable that the first stage in this process is a tendency to prolapse of the cæcum into the pelvis as a result of the upright position in patients suffering from habitual constipation.

"Bands form between the lateral abdominal wall and the colon in Nature's effort to afford support, and these, while serving their purpose to some extent, hamper the peristaltic movements of the colon, and thus increase the stasis of its contents. This condition is naturally seen more on the ascending colon than on the descending, as the hepatic flexure is much less securely supported than the

splenic flexure, and there is, owing to the force of gravity in the erect position, a greater tendency to stasis in the ascending than in the descending colon.

"The effect on the ileum is, I believe, a later development, and results from the fact that as the cæcum descends it drags upon the ileum, which has at its termination a very short mesentery. The effect of constant dragging on this mesentery causes a thickening, which eventually becomes a band kinking the ileum at its point of attachment.

"The effect on the patient's health is very insidious. Starting with the cardinal symptoms of pain in the abdomen and constipation, there are gradually added all the characteristic signs of toxæmia, *e.g.* foul tongue and breath, sallow skin, loss of weight, headache, lassitude, and so-called neurasthenic symptoms. The right kidney is often too mobile, and cystic changes occur in the breasts. These patients soon find that the recumbent position eases their pain to a marked degree, and this fact, with their general loss of tone, induces a form of chronic invalidism. In the late stages the condition is readily recognised, but in the early stages it may be easily mistaken for appendicitis."

The recognition of these cases both in their early and late stages will be fully considered in Part II.

II

In my previous paper I pointed out that there were three distinct groups of conditions that arise mainly or entirely from intestinal toxæmia. Since that paper was published, an interesting and instructive paper by Adami has appeared in the *British Medical Journal*, in which he draws attention to the important part played by a sub-infection from the intestinal bacteria in the causation of many chronic diseases. Adami is in agreement with Lane in attaching profound importance to the intestinal tract as a source of poisoning in a large and varied group of medical affections. He differs from Lane, however, in regard to the etiology of the conditions. His view, which is supported by the results of much careful experimental investigation referred to in his paper, is that the diseases in question are the result of a very slow, attenuated *infection* from the intestinal bacteria—a condition for which Adami has coined the term "sub-infection"; and in his view this sub-infection

should be differentiated from a simple toxæmia. However desirable this is from the scientific standpoint, it is not at the present time practicable nor essential. For practical purposes there is really no difference between the teaching of Lane and the teaching of Adami. Intestinal stasis, on which Lane lays so much stress, almost of necessity implies a pathogenic action of the intestinal bacteria. If any practitioner has doubt on this subject, that doubt would be set at rest by the systematic examination of the stools in a few cases of intestinal stasis. To my mind, therefore, Adami's views furnish very important and instructive evidence in favour of the general correctness of Lane's teaching. In the present stage of our knowledge the term intestinal toxæmia necessarily includes the effects of the toxins arising from the pathogenic effects of the normal intestinal bacteria, as well as those resulting from abnormal activity of the normal intestinal ferments. Keeping this definition in mind, I pointed out in my previous paper that there were three distinct groups of conditions arising mainly or entirely from intestinal toxæmia: (1) A neurasthenic group, which includes cases often regarded as neurotic, and also a sub-group in which the symptoms are mainly mental. (2) Rheumatoid arthritis. (3) A "dyspeptic" group.

The distinction between groups (1) and (3) is decidedly arbitrary, but is justified on practical grounds. The cases of neurasthenia referred to in group (1) are cases associated with well marked visceral lesions in the alimentary tract. Conversely, many of the cases referred to under the dyspeptic group present marked neurasthenic symptoms. My object in the present paper is to illustrate these different groups.

The great majority of the cases included in my series have been ill for a long time, and had previously been under prolonged medical treatment. In connection with these cases I may say that there is at the present time no type of case which presents more difficult problems in therapeutics. More particularly the difficulty arises as to the class of case calling for operative interference; and further, when operative interference appears advisable, the difficulty arises as to the exact nature of the operation. At the present time our views on both these questions must be of a tentative nature. Our knowledge of the diagnosis of these conditions is at the present time considerably ahead of our knowledge of the best means of treatment. Intestinal toxæmia, in the sense here regarded, is a common condition both in private and hospital practice. My object in this paper is

to stimulate a closer investigation of the subject, so that the general correctness or otherwise of the views here advocated may be determined. In the following records I have selected cases as types which illustrate the practical difficulties encountered in regard to the diagnosis and treatment. The general treatment of these cases will be fully detailed in my next paper.

In the first place, I will illustrate cases of neurasthenia, with general visceroptosis, and intestinal stasis, an association of conditions which has lately been receiving a good deal of attention from American physicians and surgeons.

Neurasthenia, Constipation, General Visceroptosis, Cæcal and Colon Stasis.—Mrs G., aged 34, was sent to me by Dr Veitch of Tayport, complaining of a "dead-weight feeling behind the sternum," easily tired, constipation and depression, these symptoms being of three years' duration. Mrs G. further stated that she never enjoyed her food; she felt that she was not taking food naturally. She had to lie down a little every day to get energy; and she often put up fluid from the stomach, which resembled "sour beer." In writing to me, Dr Veitch explained that the beginning of the trouble was in July 1910. "While in Oban she had a slight herpetic attack, and fainted in an hotel there. On coming home she complained of discomfort under the sternum, with flatulence after food, inclined to be tearful, and felt as if something were keeping her down." The advice of various consulting physicians had been followed without marked relief.

On objective examination the patient presented a slightly toxic appearance, with fair general nutrition. The abdomen was slightly prominent, the abdominal wall rather soft and flabby, and there was marked splashing in the epigastric region. Slight pain was complained of on deep pressure a little above and to the right of the umbilicis. The urine presented no abnormality; the stools were markedly constipated, and contained an excess of mucus. Other systems were normal. The diagnosis of neurasthenia was made, associated with constipation and general atony of the stomach and large bowel, and for six weeks she was treated along the following lines: Complete rest in bed for four weeks; a mercurial pill (Pil. hydrarg., gr. 1) given every night for a week, followed in the morning by 1 to 1½ ounces of Henry's solution. The skin was stimulated by a daily shower-bath of hot, followed by cold, water. The function of the kidneys was promoted by 12 ounces of hot water

three times a day on an empty stomach. Massage was daily applied, including vibratory massage to the abdomen and spine. For two days at the outset the diet was restricted to skim-milk and chicken-tea, with waters of toast. Thereafter nitrogenous foods were added in the form of fish, chicken, white of egg, custard, curds, and jellies, the farinaceous foods being markedly restricted for the first three weeks. After six weeks' treatment the condition of the patient was decidedly improved, there being a definite improvement in most of the symptoms, and the patient was sent away for a holiday. The relief from symptoms, however, proved to be short-lived, and in a few months the condition was as bad as ever. An X-ray examination was then made, and brought out the following points: There was a definite proptosis of the stomach, the lower curvature coming four inches below the umbilicus, the stomach emptying itself completely within six hours (Fig. 5). Examination at the end of six hours showed a distinct degree of ileal stasis, a large quantity of the meal not yet having re-entered the cæcum (Fig. 6). In 24 hours the cæcum and the first part of the ascending and transverse colon were full, the transverse colon being markedly proptosed, the first part of it lying in close relationship to the ascending colon, presenting a characteristic "shot-gun" appearance. The appearances after 48 hours revealed the ascending colon and the first part of the transverse colon in close juxtaposition, the transverse colon being markedly proptosed. The hepatic flexure was very low—considerably lower than the usual, lying below the iliac crest. At 72 hours some of the bismuth was in the rectum, much remained in the cæcum and transverse colon, there had been no evacuation (Fig. 7). An X-ray photograph of the chest revealed a condition of considerable interest; there was marked depression of the left half of the diaphragm, and a distinct degree of cardioptosis.

The X-ray findings, therefore, are a general visceroptosis with marked ileal, cæcal, and colon stasis, these being associated with a proptosis of the lower half of the diaphragm and drooping of the heart. It is probable that one of the patient's chief complaints, namely, the feeling of dead weight behind the sternum and the feeling as if the food did not go into the stomach naturally, was dependent upon some abnormal stretching of the structures associated with the general ptosis. The drooping of the left half of the diaphragm is a condition which I have not previously seen described. It is not quite clear to me how this has been brought about. As medical measures along the above lines have failed to

FIG. 5.—Mrs G. After bismuth meal. Enlarged proptosed stomach.

PLATE I.—FIG. 6.—Mrs G. Six hours after bismuth meal. Shows marked degree of ileal stasis.

FIG. 7.—Mrs G. Seventy-two hours after bismuth meal. Shows marked proptosis of transverse colon, also cæcal and general colon stasis.

PLATE II.—FIG. 8.—Miss G. Enlarged and markedly proptosed stomach.

give the patient marked and permanent relief, and as the X-rays reveal the existence of very marked stasis with visceroptosis, the advisability of operative treatment is now being considered, the operation favoured being a partial or complete colectomy.

A similar case, even more marked in degree, is an example of a type of patient who are often regarded as poor "neurotic" creatures, when in reality careful examination reveals a very definite organic basis for the trouble.

In the case in question her own medical man was alive to the important part probably played by the condition of the bowel, but the impression conveyed to the patient by the opinion of other medical men under whose care she had been had somewhat depressed her, in so far that they had appeared to consider that there was no organic basis for her complaints.

Neurasthenia, Visceroptosis, Intestinal Stasis.—Miss G., aged 27, a patient of Dr Fowler, Juniper Green, complained of a feeling of discomfort in the abdomen often amounting to pain, especially on the right side, of frequent "rumblings" in the abdomen, and general lack of vigour of over two years' duration. She also had a slight cough. The patient had been in India some months before. There had been some difficulty in arriving at a conclusion as to whether her condition of ill-health was due to some intra-abdominal condition, or to latent tuberculous lung mischief. The charts which were sent to me showed that for months there had been a slight elevation of temperature, amounting to 99° and 99.2° F., most marked in the morning. The family history showed marked tendency to nervous disorder.

On objective examination the patient was exceedingly thin, of poor nutrition, and slightly anæmic. She quite looked her description of her own condition, "as not knowing what a day's health meant," and this condition was rather aggravated by the feeling of uncertainty in her own mind as to whether there was any organic basis for her trouble, or whether it was purely "neurotic." The chest was poorly developed and somewhat flat, poor expansion at the right apex and left base, with exaggerated breath sounds at the right apex and slightly diminished over the left base; no indication of any active mischief, and no expectoration. The abdomen was extremely soft and flabby, with very pronounced splashing in the epigastric region and also in the cæcal region. There was distinct

pain on pressure in the epigastric region midway between the umbilicus and the base of the xiphisternum, and also over an area about two inches square in the right iliac region. The tongue was dry and furred, the appetite poor, flatulence pronounced, stools constipated and markedly offensive, with a trifling excess of mucus. The urine contained many catarrhal and pus cells, but it gave no growth of organisms on bacteriological examination; there was slight leucorrhœa.

On examination by the X-rays, the stomach was found to be markedly proptosed and also enlarged, the position and size being indicated in Fig. 8. Six hours after the bismuth meal there was a very small amount of food remaining in the stomach, with marked accumulation in the lower coils of the ileum, a state of typical ileal stasis being shown in Fig. 9. Twenty-four hours after, the bismuth meal filled the cæcum and transverse colon, the latter being markedly proptosed (Fig. 10); after 48 hours the condition was as shown in Fig. 11. The X-rays therefore show marked general ptosis of the stomach and bowel, with very slight gastric stasis, marked ileal stasis, and pronounced cæcal and colon stasis. The diagnosis arrived at was one of neurasthenia associated with and dependent on intestinal stasis and toxæmia. Here, again, it is impossible to say from the data available to what extent the abdominal condition was aggravated by any focal mischief in the appendix region or adhesions in the right lower quadrant of the abdomen. An interesting feature in the history was the fact that her twin sister suffered many years ago from similar symptoms, and was ultimately operated on for appendix trouble, but the patient derived no benefit from the operation.

A course of medical treatment was carried out along the following lines: Complete rest in bed; food, chiefly of a protein kind, was given in small quantities and relatively dry; five grams of urotropin were given in ten ounces of water night and morning. The bowel was emptied by an enema, followed by irrigation once daily, and a course of massage, including special vibratory massage to the abdomen, was given, the state of the stools being relied on as a guide in determining any modification in treatment. At later stages aperients were given, more especially castor-oil in two-ounce doses, every second day, as that was found to be the most efficacious and satisfactory drug for the patient. After six weeks of this treatment there was a material improvement in the stools and in the appearance and general colour of the patient, and there was more tone in

FIG. 9.—Miss G. Six hours after bismuth meal. Shows traces of food in stomach and marked ileal stasis.

PLATE III.—FIG. 10.—Miss G. Twenty-four hours after bismuth meal. Shows cæcal stasis and proptosis of, with stasis of transverse colon.

FIG. 11.—Miss G. Forty-eight hours after bismuth meal. Note marked cæcal and transverse colon stasis.

PLATE IV.—FIG. 12.—Mrs W. After bismuth meal. Note enlarged proptosed stomach.

the abdominal muscles. The patient was sent away for a change, a course of petroleum, a tablespoonful three times a day, being prescribed. Some months later I heard from her that there had been a steady gain in weight, an increase in general vigour, her condition on the whole being materially improved, although still very far short of anything approaching a normal state of health.

The next case illustrates intestinal stasis and neurasthenia associated with an extremely septic condition of the mouth. The patient had devoted considerable attention to his teeth but was the victim of very unwise dentistry. It is probable that the septic condition of his mouth had been a factor of very great importance in the etiology. According to Lane, pyorrhœa and oral sepsis are very common in cases of intestinal stasis, *as a secondary occurrence*. I do not share this view. I agree with those who hold that a septic condition of the mouth in its various forms is not infrequently the primary factor in cases of intestinal toxæmia, or, as Adami would have it, sub-infection. A vicious circle is thereby set up, with the result that removal of the primary condition in the mouth is not necessarily followed by recovery. Attention must at the same time be directed to the secondary bowel trouble, which is capable in the later stages of playing a primary *rôle*.

Neurasthenia, Constipation, General Stasis with Mucous Colitis.—Mr I., aged 40, was sent to me by Dr Mill of Leith, complaining of lack of energy, both physical and mental, insomnia, palpitations, headache, and general depression, these symptoms being of many months' duration. He had lost ten pounds in weight in the previous six months. Twelve years ago the patient had an attack of severe mucous colitis, for which he had been treated from time to time.

On objective examination the patient presented the characteristic appearances of neurasthenia. In addition to the symptoms above described, he complained of a curious shuffling feeling about the feet, and a feeling of uncertainty as to the soundness of his right side and leg. His teeth were found to be in a very unsatisfactory state, an upper plate being fixed in permanently in a very unhygienic manner, and in addition one or two of his teeth required attention.

On physical examination of the patient in the upright posture the lower part of the abdomen protruded in a manner characteristic of ptosis of the bowel. The general tone of the abdominal wall was

poor, and there was marked splashing in the epigastric region and to a less marked extent in the right iliac region, with pain on pressure in the latter region. The stools were markedly alkaline in reaction and contained much undigested food and excess of mucus. The urine gave a marked reaction for indican. On examination after a bismuth meal the stomach was found to be large and atonic; it emptied itself completely in six hours. There was distinct stasis in the terminal coils of the ileum, and pronounced cæcal and colon stasis, the cæcum, ascending, and transverse colon containing much bismuth contents at the end of 48 hours. Other systems were normal. The condition was clearly one of neurasthenia associated with and dependent on constipation, with marked atony of the whole gastro-intestinal tract, with proptosis of the transverse colon, and a mucous colitis. The only point of difficulty about the diagnosis was to determine whether or not there was a lesion in or around the appendix, or adhesions in that region involving the bowel in the neighbourhood of the ileo-cæcal valve. Any conclusion on this point was deferred until after observing the results of medical treatment. This consisted in: The correction of the state of the teeth and gums, complete rest in bed for three weeks, the administration for a few days of a very light protein dietary, daily lavage to the lower bowel, and vibratory massage to the abdomen. At the end of a fortnight systematic exercises were prescribed, calculated to strengthen the abdominal muscles, and the diet was made more liberal, special care being taken to restrict the farinaceous foods. In addition, the liver and bowels were acted on occasionally by calomel and a morning saline, and later by the administration of petroleum and tonic doses of cascara and liquorice given three times daily. When allowed up, the use of an abdominal belt was prescribed. Under this régime the patient made a very satisfactory recovery; his physical and mental vigour returned, the abdominal tenderness disappeared, the state of the stools became practically normal, and he put on six pounds in weight, this improved condition being maintained two months after his return to work.

We will now illustrate the important relationship between intestinal toxæmia and rheumatoid arthritis. A remarkable change has occurred in the views as to the etiology of this disease in the past few years, and there is now an increasing consensus of opinion among clinicians that the disease is of infective origin the infection in the great majority of cases being intestinal in origin. I fully share this

view, and am satisfied from close observation of a large number of cases that the poison or poisons that are responsible for the disease are absorbed into the system largely through the digestive tract. In some cases the derangement of the bowel present is apparently secondary to some sepsis higher up, more especially pyorrhœa alveolaris and septic conditions of the teeth. In other cases the catarrhal condition of the bowel or the marked intestinal stasis is a primary affection. A vicious circle is set up, which in turn aggravates the condition. In my former paper I gave expression to the opinion that different bacteria may induce the pathological pictures comprised under the term rheumatoid arthritis. If that is so, it indicates that, in addition to a bacterial factor, there are other important factors at work, which is not unlikely. Let me cite shortly one or two illustrative cases which have come under my own observation.

Miss M., aged 25, was sent to me by Dr Robertson of Bonnyrigg in September 1912 suffering from well-marked rheumatoid arthritis of three years' duration. The small joints of the hands, also the ankles, back, and occasionally the jaw, were all involved. When seven years old she had a very bad attack of stomach trouble, from which she was not expected to recover.

On objective examination, in addition to the fusiform, painful enlargement of many of the joints referred to, it was found that the urine contained numerous catarrhal cells and white blood corpuscles, and the bacteriological examination of a catheter specimen of urine revealed on two occasions a few colonies of staphylococcus albus. The stools were constipated and offensive and remained so for some weeks. In the course of treatment directed to improve the condition of the stools the patient developed a slight attack of appendicitis, and in a few days, when the attack had abated, it was resolved to remove the appendix. At the operation Mr Scott Carmichael found the appendix showing evidence of a recent catarrhal appendicitis; bacteriological examination of the muco-pus present in the distal end of the appendix revealed the presence of staphylococci, from which a vaccine was prepared for use later. A month after recovery from the operation the pains in the joints were still present, though in less degree, and the stools were by no means normal. The administration of an autogenous vaccine was then begun and continued weekly for three months. The result of treatment was satisfactory in every respect, and for the past year the patient has

virtually been free from any symptoms, having made practically a complete recovery from the disease.

The following case illustrates the marked benefit that may follow the operation of short-circuiting (ileo-sigmoidostomy) in advanced cases:—

Mrs P., aged 52, complained of pain in the left thigh, and of painful enlargement of the joints of the hands and fingers of about three years' duration. Four years ago she had an attack of severe appendix trouble, for which an operation was at one time deemed advisable, but this was not carried out. She has had a considerable amount of digestive disturbance in the past three years. The rheumatism was now beginning to cripple her movements seriously. She had taken veronal every second or third night for over ten months.

On objective examination the patient presented the typical appearances of early rheumatoid arthritis, with a considerable amount of fibro-myositis in the left thigh. The urine contained many pus cells, and a bacteriological examination of a catheter specimen revealed the presence of staphylococcus albus. The stools were very constipated and offensive, containing much excess of mucus. A considerable degree of discomfort was complained of in the right side of the abdomen. A course of medical treatment carried out for six weeks failed to give any material relief of the symptoms, or to markedly improve the condition of the stools, and as X ray examination after a bismuth meal revealed the presence of very marked cæcal and colon stasis, after consultation with Professor Alexis Thomson, the operation of short-circuiting (ileo-sigmoidostomy) was decided on, and this was carried out. In the third week after the operation acute obstruction set in, due to the presence of adhesions, which were relieved by further operation. Subsequent convalescence was retarded by the development of cystitis through catheter infection, but when that was overcome the patient made a very satisfactory recovery. The relief from her rheumatic symptoms was most marked; her general appearance and sense of vigour improved enormously, and for the past eight months there has been no question of sleeping powders. The one untoward result of the operation, however, is that the bowels move four or even five times daily. This is a disadvantage, as it curtails the patient's freedom of movement.

This brings me to the illustration of the third or "dyspeptic" group of cases. The first case affords a proof of the statement that in the later stages of this condition the diagnosis is a relatively easy matter, if recourse is had to radiography, but the results of treatment at the present time are more doubtful.

Chronic Indigestion, Increasing Debility, with Intestinal Stasis.—Mrs W., a patient of Dr Cullen of St Boswells, complained of recurring bilious attacks, with flatulence, marked loss of vigour, severe headaches, nausea and aversion to food, and constipation, these symptoms dating from an acute attack of sickness and vomiting in 1910, her general condition having become decidedly worse since an attack of shingles a few months ago. The patient was never strong, and was always of a nervous disposition. She was troubled occasionally by marked frequency of micturition, her weight, 7 st. 12 lbs., was a stone less than her normal.

Objectively the patient was in poor condition, her nutrition was very poor, and she was decidedly anæmic. On physical examination the abdomen was extremely flabby, and there was marked splashing in the epigastric and iliac regions, with an area of tenderness in the right iliac region, where the patient sometimes complained of a dragging sensation. There was a slight degree of sepsis in connection with one tooth. The urine contained many catarrhal cells and pus cells, and was specially prone to bacterial decomposition; the bacteriological examination of a catheter specimen of urine gave a negative result. The examination of the stools showed them to be abnormally offensive, the irrigating fluid which was used immediately after clearing out the bowels by an enema returning with much feculent and offensive matter, with excess of mucus.

An X-ray examination after a bismuth meal showed the stomach to be slightly enlarged, proptosed, and atonic (Fig. 12), and that it failed to empty itself within six hours. At this period a bismuth shadow was detected in the first part of the duodenum (Fig. 13), indicating that there was some interference with the free passage of food through it. There was marked accumulation of bismuth contents in the terminal coils of the ileum at this time (Fig. 13). Later, marked cæcal and colon stasis was determined, a considerable amount of the meal being present in the colon and some in the cæcum at the end of 72 hours. The diagnosis arrived at was intestinal stasis, the primary lesion being in the terminal part of the ileum, possibly associated with adhesions. A course of medical treatment was

decided on and was carried out for fully six weeks. The amount of benefit that followed thus was practically negligible, it being apparent that the slight benefit experienced by the patient was immaterial, and would soon disappear when she resumed her household duties. An operation was recommended, and after consultation with Professor Alexis Thomson, the operation of the ileo-sigmoidostomy was decided on. I am indebted to Mr Thomson for the following note of the surgical findings: Incision of right of middle line; the absence of parietal fat was noted. The stomach was normal, except that the pyloric sphincter was contracted in so far as it resisted the passage of the index finger. The duodenum in its second and third portions was considerably dilated, so that the contrast with the jejunum was very marked. The cæcum was devoid of a mesentery and capable of abnormal range of movement; the appendix showed no trace of previous mischief; the ascending colon was crossed by cicatricial bands, which narrowed it and bound it to the adjacent right half of the transverse colon, the latter being dragged down so as to be nearly parallel with the ascending portion, with a corresponding diminution of the angle of the hepatic flexure. The lower end of the ileum was fixed by cicatricial bands related to its mesentery, so that it tended to be kinked about three or four inches from the cæcum—between the kink and the cæcum it was decidedly narrowed, partly by the cicatricial bands and partly by contraction of the circular muscular coat. The pelvic colon was small and empty and its mesentery so short that the loop could not be presented at the wound. The uterus was enlarged by intramural fibroids. The gall-bladder showed no trace of previous trouble.

The cicatricial bands in relation to the ileum and ascending colon were divided, the raw surfaces that resulted from this were covered with adjacent peritoneum, and the cæcum was anchored by the method of Wilms. All peritoneal surfaces in affected areas were smeared with vaseline.

This case is interesting as showing the striking value of the X-rays in the diagnosis of such conditions—the cæcal and colon stasis, the ileal stasis, the duodenal lesion, and the gastric stasis found at the operation having been previously determined by the X-rays.

In view of the severity of the adhesions involving the terminal portion of the ileum, cæcum, and ascending colon it was thought

probable that treatment of the adhesions coupled with the anchoring of the cæcum above described would suffice to benefit the patient considerably. With regard to the result of the operation, sufficient time has not yet elapsed to allow of any opinion being given, but our initial impressions six weeks after the operation are decidedly unfavourable, and should these impressions be borne out by subsequent events, the result will justify the view that the various adhesions to which attention was mainly directed at the operation are a result and not a cause, and that more radical surgical measures will be necessary if the patient is to be materially benefited.

In my former paper I mentioned the present tendency to regard various duodenal and gastric conditions as secondary and not primary affections. In recent years the experimental work of Wilkie and others has clearly proved the existence of an ascending infection from the ileo-cæcal region. The following is a good example :—

Duodenal Ulcer following Appendicitis, and Cæcum Mobile associated with Intestinal Stasis.—Mr S., aged 36, complained of recurring indigestion of many years' duration. For the past two years attacks of sickness with vomiting have occurred with increading frequency, these being followed by periods of relief lasting for a few days up to many weeks. He had long suffered from persistent constipation and general want of tone. Previous history unimportant, except for an attack of inflammation of the bowels when about fourteen years of age, the history suggesting appendicitis.

On objective examination the patient was a spare, somewhat toxic-looking man. The stomach was slightly dilated, and there was marked splashing in the cæcal region. There was slight tenderness on pressure over the region of M'Burney's point and in the epigastric region.

On examination after a bismuth meal the stomach was found to be in a markedly hypertonic condition, the bismuth being seen to rush through the duodenum with great rapidity. At the end of six hours the stomach was empty, the bismuth contents being almost entirely in the cæcum ; there was no ileal stasis. After 48 hours the cæcum, ascending and transverse colon, were still full of the bismuth contents, the bismuth meal not being entirely voided after 80 hours.

A course of medical treatment was tried for about a year, the patient, however, never being laid up. After careful dieting and the administration of antacid powders a very marked degree of relief

was obtained for about eight months. This relief, however, was incomplete, and at the end of a year the symptoms were as bad as ever. After consultation with Mr Wade, an operation was decided on which would allow of free examination of the appendix and the duodenum. I am indebted to Mr Wade for the following note of the operation:—

(Anæsthetics—morphine, one-sixth; atropine, $\frac{1}{100}$, half an hour before the operation. Novocaine, quinine, urea, gas, and oxygen.)

A long incision was made through the middle of the right rectus muscle, through which the abdomen was opened. The omentum was found matted over the cæcum and adherent to the appendix, which was recto-cæcal in situation. A characteristic Lane's ileal kink was found. The duodenum was dilated, and a circular duodenal ulcer was present on the anterior wall of its first part. The band of tissue uniting the ileum to the right wall of the pelvis that produced the ileal kink was divided. The appendix was then dissected out and found to be bulbous at its tip, and matted by inflammatory adhesions as from a former appendicitis. The appendix was amputated and the omental adhesions divided. The cæcum was then plicated and fixed to the abdominal wall. The duodenal ulcer was then carefully re-inspected, and it was decided that it was not necessary to do a gastro-enterostomy. An omental graft was, however, brought down over its serous surface and united to the duodenum.

Progress Notes. No post-anæsthetic sickness. The abdominal discomfort due to slight meteorism was relieved by heroin. Aperient given on the third day; clips removed on the sixth; patient out of bed on the tenth day, and home in the middle of the third week.

The next case is a particularly interesting one, as it illustrates the toxæmic or infective origin of rheumatism. It is further a typical example of what Sir Arbuthnot Lane describes as the 'static' variety of intestinal stasis.

Rheumatism. Intestinal Adhesions and Kinks. Intestinal Stasis of the Static Variety. Miss M., aged 40, consulted me in September 1913 on account of rheumatism in the neck, general lack of vigour, and indigestion of over nine months' duration. Previous history was unimportant.

On objective examination the patient's general appearance was unsatisfactory, the complexion being sallow, showing evidence of slight anæmia; the tongue was furred; there were numerous areas

of inflammatory thickening of the muscles of the neck (fibro-myositis). Some of the teeth were in an unsatisfactory state. Examination of the abdomen revealed nothing abnormal; the bowels were stated to be regular. Other systems were normal. Some idea of the thoroughly unsatisfactory state of health may be learned from the later history. The day after she took to bed she passed into a condition of acute delirium, necessitating a special nurse, night and day, for four dyas. This attack was associated with slight fever, and was obviously toxic in origin. A week later she developed acute tonsilitis, followed by abscess formation, which was followed in turn by acute middle-ear catarrh. After recovery from this she developed extensive and severe acne of a somewhat suppurating type, and on recovery from this she developed an acute attack of pain in the right lower quadrant of the abdomen, accompanied by fever and some localised tenderness, the condition presenting all the clinical features of an attack of appendix trouble. This history shows the extremely unsatisfactory and septic state of the patient's tissues, and it was clear that the " rheumatism " originally complained of was merely a clinical manifestation of the septic condition. After the teeth had been attended to, a course of medical treatment was prescribed, this including complete rest, light diet, massage and baths, with aperient drugs. A closer inquiry into the history had revealed the existence of very definite constipation, the existence of which at first was not appreciated by the patient. After prolonged treatment the general condition of the patient improved markedly ; the fibro-myositis was almost entirely got rid of, her sense of vigour was improved to some extent, and her digestion was on the whole satisfactory, and she was sent away to convalesce in the country for six weeks, in the hope that her vigour would materially improve, and that she would put on some weight. These hopes were not realised. After living under very favourable conditions in the country for six weeks the weight had not been added to, there was no material gain in vigour, and her indigestion had been worse, pain having been frequently complained of, necessitating her lying down for a few hours on end. On examination there was an area of tenderness on pressure in the right iliac region, the stools were constipated and were unduly offensive. The conclusion was arrived at that in all probability the *fons et origo* of all her troubles was to be found in the ileo-cæcal region, arising mainly from long-standing constipation, the local tenderness on pressure further suggesting some chronic appendicular mischief, and, after consultation

with Mr Wilkie, operative measures were decided on. I am indebted to Mr Wilkie for the following note: "Right paramesial incision 5½ inches long, centre opposite umbilicus. On opening the peritoneal cavity a long, lax transverse colon presented. On lifting this up there were seen to be numerous adhesions stretching from the transverse meso-colon to the first coil of jejunum, and to the mesentery of the upper part of small intestine. The uppermost jejunal coil was larger and felt somewhat thicker walled than the normal. At the duodeno-jejunal junction the jejunum turned sharply to the right, where it was held up to the transverse meso-colon for a distance of about three inches. These adhesions were divided with scissors, and the raw surface on the wall of the jejunum covered over by a continuous Lembert suture. The stomach was somewhat proptosed and was flabby, but showed no external evidence of ulcer. The first portion of the duodenum appeared normal it was not dilated. The third part of the duodenum appeared dilated, but its wall was not hypertrophied. The terminal coil of the ileum was free and lax and showed no evidence of dilatation nor hypertrophy. The appendix was somewhat thicker than normal in its distal half, and was fixed in its middle portion by an adhesion in the pelvis. On cutting this the appendix was brought up and removed. It contained foul-smelling feculent material. The cæcum was slightly dilated, but was not unduly mobile; its upper part was covered by a typical Jackson's membrane. In the region of the ascending colon there were several stellate fibrous adhesions stretching from the flank across the front of the colon. These were divided by freeing the ascending colon. The transverse colon in its proximal two inches was fixed to the ascending colon, causing a somewhat sharp angulation at the hepatic flexure. The connection between the two loops was severed; there was no similar adhesion at the splenic flexure. The pelvic colon was not bound down, but there were some white cicatrices in its mesentery. The abdomen was closed in layers by catgut sutures, and the skin wound by Michel's clips and silkworm gut sutures."

Summary of Positive Findings.—(1) Jejunal angulation with adhesions to meso-colon; 2° appendix kinked by adhesions; (3) well-marked pericolic bands over ascending colon. Some angulation at hepatic flexure.

Too short a time has elapsed since the operation to allow of any statement being made as regards the result, but eight weeks after

FIG. 13.—Mrs W. Six hours after bismuth meal. Note bismuth shadow in stomach (gastric stasis), duodenum, and ileum (marked ileal stasis).

PLATE V.—FIG. 14.—Mr H. Six hours after bismuth meal. Note bismuth shadow in stomach (gastric stasis) and ileum (ileal stasis).

the operation the patient expresses herself as feeling better than she has felt for years ; she certainly looks immensely improved, and the initial results from the operation are very promising.

Chronic Disease of Appendix, Secondary Duodenal Ulcer, Intestinal Stasis.—Mr H., aged 38, was sent to me by Dr John M'Laren, complaining of pain and discomfort in the right side of the abdomen, and feeling of unfitness for work on account of physical and mental depression, and having lost three stone in weight in the past three years. The illness began four years ago with a sharp pain in the pit of the stomach, which doubled him up for about two hours. About three years ago pain set in in the right side, and had been more or less present ever since. For the past three years the bowels had been markedly constipated. Various methods of treatment had been tried by Dr M'Laren, with expert assistance, without material benefit.

On objective examination the general appearance of the patient was that of a poorly-nourished, rather anæmic and ill looking man. His teeth were not altogether in a very satisfactory state. On examination of the abdomen there was marked pain on pressure below the xiphisternum, and also in the right iliac region, pain on pressure in the latter area occasionally setting up acute epigastric pain. The urine contained many catarrhal cells, white blood corpuscles, and a few red blood-cells.

On examination after a bismuth meal the stomach was found to be somewhat large, a little displaced downwards, and in a markedly hypertonic condition. At the end of six hours a fair amount of bismuth meal was still present in the stomach (Fig. 14). Much of it was present in the terminal coils of the ileum, the great bulk of it being in the cæcum, ascending colon, and first part of the transverse colon (Fig. 14). After 24 hours the bismuth meal was still in the cæcum, ascending and transverse colon, the ascending colon being somewhat dilated (Fig. 15). After 48 hours the bismuth contents were present in the splenic flexure, descending colon, and rectum, and the cæcum was empty ; there was still, however, marked delay at the hepatic flexure and onwards. The X-ray appearances, therefore, proved the existence of gastric stasis, ileal stasis, and cæcal and colon stasis, the further questions remaining were whether this stasis is of a static variety or is associated with disease in the appendix and marked adhesions, with or without secondary duodenal mischief. The *tout ensemble* of the clinical

history, with findings on objective examination, rather points to chronic disease of the appendix, associated with secondary gastro-duodenal mischief (ulcer with spasm of pylorus). Further observation and investigation are necessary to determine this point, and this will be done before operative measures are decided on.

The above cases are typical examples of moderately severe forms of intestinal stasis with its associated toxæmia. As I have already indicated, it is impossible to differentiate clinically, cases of toxæmia in the sense of Lane, and cases of sub-infection in the sense of Adami. I have seen several examples which serve admirably to connect the two groups, and also illustrate the advantages of plication and fixation of the cæcum in cases of enlarged and mobile cæcum.

A girl of 15, a patient of Dr John Playfair, suffered from constipation and frequency of micturition for some years. Since the age of four or five she had never been strong. Before she came under my observation she had been staying a short time in England where she was seriously ill from a severe febrile attack of two or three weeks' duration. The patient was a pale, pasty looking girl of moderate nutrition. The urine contained a few catarrhal and pus cells and on bacteriological examination showed a pure growth of B. coli. X-ray examination revealed marked ileal, cæcal and colon stasis the cæcum being notably enlarged and displaced downwards. There was slight pain on pressure in a localised area in the right iliac region. The question arose as to whether, in view of the definite positive findings, it would be worth while to try a course of medical treatment, supplemented by vaccine therapy, but after consideration this course was not thought advisable, and after consultation with Mr Wade an operation was decided on. The operation fully confirmed the X-ray findings. The cæcum was plicated and stitched to the abdominal wall, and the appendix which appeared slightly thickened and adherent, was removed. The patient made a very satisfactory recovery after the operation, and has been immensely benefited by it. In a few weeks the urine cleared up entirely and the frequency disappeared. The photograph (Fig. 16) shows the cæcum, ascending colon, and the terminal coils of ileum; the three marks in line are placed over the abdominal cicatrix.

I refer specially to this case as it is a good example of several

FIG. 15.—Mr H. Twenty-four hours after bismuth meal. Note dilated condition of ascending colon, with slight stasis.

PLATE VI.—FIG. 16.—Girl æt. 15. Six hours after bismuth meal. The three small black marks are over the cicatrix.

FIG. 17.—Six hours after bismuth meal (recumbent posture). Note the prominent ileal stasis and slight dilatation of the recently narrowed cæcum. Three small black marks are over cicatrix.

PLATE VII.—FIG. 18.—Miss A. Thirty-two hours after bismuth meal. Note the double looped appearance of the elongated and dilated transverse colon in a state of stasis.

for which the same operation has been done by Mr Wade for me with marked benefit. I should refer, however, to one precaution in regard to it, and illustrate it by reference to Fig. 17.

This patient had suffered from persistent bad health for a long time, and various methods of treatment had been tried without marked benefit. The case had been regarded as one of gall-stones. Examination of the case showed it to be a typical one of intestinal stasis, probably associated with and aggravated by some old appendix mischief, and she was operated on by Mr Wade, the cæcum being plicated and stitched to the abdominal wall, and the appendix, which was the seat of some adhesions, was removed. The patient made a very satisfactory recovery, and at the end of six weeks was in better health than she had been for a very long time. One day at this time she strained herself lifting a heavy table, which brought on a severe pain in the right iliac region, with marked localised tenderness suggestive of some local inflammatory mischief; this disappeared slowly. A month later an X-ray photograph was taken, and showed the interesting condition shown in Fig. 17. This is taken in the recumbent posture six hours after a test meal. It shows very pronounced ileal stasis, and it also shows a dilated condition of the cæcum, which strongly suggests that some of the stitches had given way under the influence of the strain. It will be interesting to watch the later results of this case.

In my former paper I mentioned that most of the X-ray photographs had been taken in the vertical posture. It is important to control the results in many cases by examining in the recumbent position. This is well illustrated by reference to Fig. 18, taken from a lady suffering from advanced intestinal stasis, which had produced a serious condition of ill-health. The degree of stasis at the end of 32 hours is well shown by the condition of the transverse colon, which shows a characteristic double loop. This double loop, pointing to a greatly elongated colon, was not visible on examination in the erect posture. This case is of very special interest, as it is one of the few cases that I have come across which has been profoundly benefited by means of diet, the beneficial effect of diet on this stasis, clinically, at any rate, being altogether remarkable. Further reference to this will be made when dealing with the more general details of treatment in my next paper.

III

Treatment

Before entering on a discussion of the principles of treatment and the details of its practice, it may be useful if I summarise the leading points dealt with in my earlier papers bearing on the question of treatment.

The subjective symptoms of intestinal toxæmia were described as of varied character, falling into three main groups—a group in which the symptoms are mainly those of neurasthenia, a second, in which the symptoms fall into the category of chronic rheumatism or rheumatoid arthritis, and a third, in which the symptoms are mainly digestive, and for convenience described as a "dyspeptic" group. In dealing with the objective appearances, stress was laid on the importance of careful and oft-repeated examination of the stools for evidence of long-standing excessive putrefaction with or without definite indications of catarrh of the bowel; also on the importance of the minute examination of the urine including bacteriological investigation, and lastly, on the great value of the X-rays in supplementing the information obtainable by the older methods of examination, the X-rays being of special value in determining the site of the stasis, and also its severity. Attention was also directed to—and too much emphasis cannot be laid on this point—the importance for looking for other sources of toxæmia which may be operative, the more important of these being some septic trouble in the mouth, associated with septic teeth and an infected state of the gums (pyorrhœa alveolaris), the tonsils, and in women, a latent uterine catarrh.

From a therapeutic standpoint cases of intestinal toxæmia may be roughly classified into three main groups—Group 1, cases in which the symptoms and physical signs are comparatively trivial, and which yield readily to simple medical treatment. Group 2, In which the symptoms are more pronounced and are accompanied by marked objective indications of an abnormal state of the digestive tract, but which are amenable to careful and prolonged medical treatment. Group 3, In which the condition of the digestive tract is so abnormal that medical measures fail to relieve, relief being obtainable by surgical treatment.

Cases which fall under one or other of these groups are met with very commonly in the everyday practice of the physician.

and I am satisfied that they form a large share of the cases which come under the daily observation of the practitioner. Many factors have to be considered in the treatment of these cases, but the keynote of successful treatment is undoubtedly to be found in the application of antiseptic or aseptic principles to practical medicine.

In a recent discussion before the Harveian Society of London, Dr William Hunter pointed out that a great desideratum at the present time is a development of an era of antiseptic medicine analogous to the brilliant era of antiseptic surgery of the last few decades. I entirely concur in this view. The whole principles of treatment and the details of its practice hereafter given are based on this principle. While this is so, I do not for a moment wish to under-rate the importance of attention to other fundamental points, such as faulty metabolism due to disturbances of chemical processes induced by agencies other than bacterial, nor to the importance of the influence of hereditary traits.

The aim of treatment in every case may be summarised as follows: (1) To correct the abnormal bacterial activity in the digestive tract, attention being directed both to the small and large intestine. (2) To correct any associated or, it may be, primary toxæmia existing in the upper part of the digestive tract, more especially the teeth, tonsils, gums, and stomach. (3) To increase the functional activity of the main channels of elimination of the natural poisons of the body, which in the cases in question are usually produced in excess; I refer more particularly to the bowels, kidneys, and skin. (4) To raise the general tissue resistance of the body by general means, attention being specially directed to improving the tone of the central nervous system. (5) In appropriate cases endeavour to raise the resisting power of the tissues by the use of vaccines.

With these introductory remarks we may pass to the consideration of the treatment in detail, and we will first discuss the influence of rest.

1. *Rest.*—Complete rest in bed is essential in the treatment of the majority of pronounced cases. In slight cases rest is unnecessary and inadvisable. Great care is called for in the selection of cases requiring complete rest. In focussing our attention in the *treatment of the intestine* we must not overlook the *treatment of the individual.* Success in the treatment of many of these cases is more easily obtained if a broad outlook on the patient's condition is taken

and the value of psycho-therapeutic influences recognised. Minor cases of intestinal toxæmia form a large proportion of the cases which derive so much benefit from a course of spa treatment. There is no class of case in which there is a greater liability for the patients to become morbidly introspective as regards their physical condition. I have on several occasions been consulted by patients who have suffered from intestinal toxæmia for prolonged periods, who came to me with a ready-made and correct diagnosis, and with a chip box full of fæces. The thoroughness which such patients show in the description of their symptoms, and the desire, shown as above, to make the physician familiar with every detail, clearly indicates a morbid condition of the nervous system which it is important to recognise and treat. In such cases treatment must be skilfully devised so as to distract as far as possible the patient's attention from his malady, and in these patients it is usually inadvisable to recommend complete rest.

Leaving this particular group aside, too much stress cannot be laid upon the value of a period of complete rest in bed at the outset of treatment in the cases in groups 2 and 3. Complete rest is valuable because the other measures to be hereafter detailed produce satisfactory results more quickly if the patient is recumbent than if going about; the favourable results of rest are also doubtless in part due to the more favourable position of the viscera in cases associated with marked proptosis. It is a wise rule to give the patient at the outset some idea of the details and duration of his treatment, this period being as a rule not less than three to four weeks. The next point to be considered is the important one of diet.

2. *Diet.*—Diet is of great value in the treatment of cases in groups 1 and 2. The explanation for this is probably in great part to be found in the influence of diet in modifying the bacterial flora in the bowel. The experiments of Herter and Kendall on the influence of dietary alternations on the types of intestinal flora are of great interest. Experimenting with monkeys and cats, these observers showed that on a protein diet the bacterial flora was of a strongly proteolytic character, but that on a change to a carbohydrate diet these organisms were rapidly replaced by others of a non-proteolytic type. Associated with this change there was a change in the putrefactive products of the urine and fæces, and a reduction in the indol, skatol, phenol, and sulphuretted hydrogen, and diminution in the indican in the urine. These observations are

of great value, as from analogy there is every likelihood that similar results hold good for man.

The toxic products produced as a result of *fermentation of carbohydrate foods* are chiefly carbonic acid and various organic acids such as lactic acid, acetic acid, butyric acid, and to a less extent formic and valerianic acid and acetone. The end products of decomposition of carbohydrate foods are carbonic acid and water, and no fermentation products are recovered from the urine with the exception of oxalic acid in oxaluria.

The toxic products arising from the *abnormal decomposition of the proteins* under the influence of bacterial action include various nitrogen and sulphur compounds, chiefly of the aromatic series, which are eliminated in the urine as ethereal sulphates. There is good reason to believe that other toxic products are absorbed from the bowel in these cases, but we have at present little knowledge of their nature.

I make a point in practice of, so far as possible, getting a correct statement of the average dietary of the individual, not only during his illness, but prior to the development of the symptoms. Two common defects are frequently found. There is one type of patient whose diet has contained an excessive amount of meat, as, for instance, the city man, who has ham and eggs for breakfast, a rich lunch in the middle of the day, and a good dinner with accompaniments at night. The converse side of the picture is met with, more especially in women, whose diet contains too little animal food, and consists too largely of bread and tea. In both these groups of cases diet is of the first importance.

The fermentation cases should be treated with a diet chiefly protein in character. The diet should comprise various meat soups, fish, chicken, eggs, tripe, sweetbread, and scraped beef, farinaceous foods being restricted to dry toast, stale bread, and rusks. The diet must be carefully planned, the food given at regular intervals, and no food of any kind given between meals. The free use of plain water or other diluent should be encouraged. It is necessary to point out the importance of proper attention being paid to mastication of the food, to the state of the teeth, and the careful regulation of the bowels, as essential points in the treatment. The following dietary is appropriate to cases of moderately severe toxæmia resulting from the abnormal fermentation of carbohydrate foods :—

7 A.M.—Tumblerful of hot water.

8 A.M. *Breakfast.*—Tea, 1 cup without sugar; milk, not cream. Good helping of fried bacon or fish, or eggs (plain poached or scrambled), or cold ham, cold tongue, or grilled kidney. One slice of thin, crisp toast with very little butter.

1 P.M. *Lunch.*—(*a*) Fish, or chicken, or game; chop, steak, roast beef, or roast mutton. (*b*) Biscuits, or dry roll with cheese, and with butter; cup of coffee.

7 P.M. *Dinner.*—Clear soup, unthickened. Meat as at lunch, without vegetables, dry toast, and a simple gravy. Pudding in form of custard, curds, jelly, cream, or stewed fruit without added sugar; no coffee.

10 P.M.—Drink of hot water.

The putrefactive cases are treated with a lacto-vegetarian diet. Meat, and especially red meats, are for the time cut off from the dietary. Meat soups are similarly withheld. Eggs are also excluded.

The diet should comprise bread and milk, farinaceous foods, green vegetables, and fruits. An appropriate diet sheet is here given—

7.30 A.M.—Tumblerful of hot water.

8 A.M. *Breakfast.*—Cereal, *e.g.* porridge, hominy, Quaker oats with cream. Toast or roll with butter, and a little marmalade if desired. One cup of tea.

1 P.M. *Lunch.*—Eggs in some form, or vegetable savoury, *e.g.* cauliflower au gratin, macaroni cheese, tomato savoury. Plain milk pudding with cream.

7 P.M. *Dinner.*—Vegetable or milk stock soup, *e.g.* tomato, artichoke, potato, lentil, or onion. Fish occasionally, or egg in some form if not taken at lunch, or vegetable savoury. Pudding—simple pudding or stewed fruit; no savouries, and no coffee.

10 P.M.—Tumblerful of hot water.

Foods Forbidden in all Cases.—Patients who are specially prone to intestinal auto-intoxication have a relatively weak digestive power. Their diet must therefore be a simple one. All foods which are difficult to digest should be excluded from the dietary, and the various "extras" which can be indulged in with impunity by healthy subjects must be rigorously withheld. The following articles should only be given in the most restricted amounts: Marmalade, jams, sugar, sweets and confectionery, pastry, sweet cakes, new bread and hot rolls, made-up dishes of all kinds, and all canned foods, pickles, sauces, spices, caviare and rich gravies, lobsters, mushrooms, shrimps, sardines, salmon, mackerel, raisins, nuts, pears, and all preserved fruits.

At the outset of treatment I frequently find it advisable to put the patient on an extremely restricted diet. I am satisfied that a policy of partial starvation for 24 or 48 hours is often of distinct value in improving the local condition in the digestive tract. Time and again I have seen cases respond very well to medical treatment where the patient lost 3 lbs. or more in the first week, and one had difficulty in satisfying the doctor in charge that the form of dietetic treatment employed was rational and sound, this starvation régime consisting of plain water, a little albumin water, skimmed milk, with possibly a few fingers of baked toast in the 24 hours. Later, in some of these cases, I find it useful to adopt a modified Salisbury régime, such as the following :—

7 A.M.—Half pint or more hot water.

8.30 A.M.—Four to six ounces meat rissoles. Two Kalari biscuits, with a little butter. Small cup very weak tea.

11.30 A.M.—Half pint or more of hot water, flavoured with lemon if desired.

1 P.M.—Breakfastcupful of beef-tea with ¼ lb. scraped meat. Thin slice baked bread or dinner toast. Half a dozen oysters (broiled). Consommé with custard, or with egg, with baked bread or toast.

3 P.M.—Half pint or more hot water.

4.30 P.M.—Breakfastcupful of skimmed milk, to which is added a full tablespoonful of Carnine Lefranc or other meat juice, or plain egg-flip flavoured with cinnamon in place of sugar. Unsweetened rusk or crisp biscuits.

6 P.M.—Half pint hot water.

7.30 P.M.—Four to six ounces meat rissoles or mince, followed by an egg jelly in which the sugar is reduced in amount, or a blancmange made with milk in place of cream.

The above régime should be maintained for 10 days or a fortnight, after which additions in the form of steamed fish, chicken, malted breads, green vegetables, jellies, and fruit can be slowly taken. The return to ordinary farinaceous foods must be made very gradually, preferably starting with the invalid foods.

In my last paper I referred to one case of severe and long-standing intestinal toxæmia with stasis of a nature which appeared to demand operation, in which a remarkably satisfactory result has been obtained by the use of the following dietary : 7.30 A.M.—Small cup of milk tea (6 ozs.). 8.30 A.M.—Cup of Benger (6 ozs.). 11.30 A.M.—Two raw eggs. 1.30 P.M.—Scraped beef-mince or

rissoles. Pudding taken from sweet jelly or curds; puree of fruit or meringue. 4.30 P.M.—Cup of milk tea and one raw egg. 8 P.M. —Cup of Benger or raw beef soup (6 ozs.). 9.30 P.M.—Small cup Horlick's malted milk or peptonised cocoa (6 ozs.).

On this diet the patient has attained a degree of comfort and vigour that she has not known for years, and, more wonderful to relate, since its adoption the bowels have moved with a regularity previously unheard of.

3. *Treatment of the Bowels.* In the mild cases all that is required is a small dose of calomel—½ to 1 grain each night for 3 or 4 nights—followed by a morning saline given in amount sufficient to empty the bowel thoroughly, the bowels being thereafter regulated by adding a little fruit to the dietary, and by including, where possible, oatmeal, whole wheat bread, and vegetables; the drinking of plain water in the morning, and, when necessary, the regular use of such aperient as is found to suit the patient. In my experience of these remedies the one I have found most generally useful is the old-fashioned senna pods used in the form of a cold infusion. It is my practice to recommend all such cases to have the functional activity of the bowel and liver increased once a week by a good dose of calomel, followed in the morning by a double-strong seidlitz powder or other saline.

It is with regard to group 2, however, that a greater difficulty is experienced in the treatment of the bowels, these being cases where the abnormal putrefaction in the bowel is pronounced, and is associated with the absorption of toxins through a diseased mucous membrane. In these cases we have—to use the language of a late distinguished clinician—to "appeal to the bowel from below" by means of enemata and irrigation. Here, as in other directions, general lines only can be indicated, great attention to detail being necessary with regard to the amount of the enema and the irrigating fluid, the results being studied as a guide in the course of treatment.

My usual practice is to give an enema of 2½ pints of soap and water, care being taken that too much alkali in the form of soap is not used, and after a preliminary clearing of the bowel by this means, to follow within 15 minutes with the introduction of from 2 to 3 pints of plain water; this is retained for a few minutes and then voided, the nature of the returned fluid giving a very important indication of the state of the bowel. In health this fluid should be returned clear, or, at most, with traces of mucus; in cases of intestinal toxæmia the returned fluid is feculent, offensive, and con-

tains mucus in great excess, this condition frequently persisting in spite of prolonged treatment. The use of intestinal lavage should be gradually reduced, a suitable aperient being then recommended for regular use. A word of caution is necessary with regard to the continued use of enemata. There is no doubt that in a certain proportion of cases the use of an enema is followed by some irritation in the appendix and cæcal regions, this being probably brought about by active reverse peristalsis in the large bowel. In my experience this only obtains in cases where there is definite local mischief in the appendix, and indicates the advisability of surgical treatment, including the removal of the appendix.

The use of petroleum or liquid paraffin is of great value as a mechanical lubricant in many of these cases. It should be given in doses of 3 to 4 drachms three times a day after food. There are many preparations in the market. I am not convinced that any of the more expensive preparations have any special value over the officinal preparation. In a very small proportion of cases I find that petroleum in any form is not well tolerated. In a few patients I have found Angier's petroleum emulsion specially useful where other preparations have disagreed.

Reference may be made to the value of different preparations of lactic acid bacilli as a means of directly neutralising the pathogenic action of the pathogenic intestinal bacteria in cases of toxæmia. Originally introduced by Metchnikoff, this line of treatment has not fulfilled expectations. On theoretical grounds much might be looked for from it, but in practice it has apparently been found impossible to put upon the market preparations which retain their active properties. The difficulties were well illustrated by me a few years ago in a demonstration which I gave to the Edinburgh Medico-Chirurgical Society on the various lactic acid preparations then in common use. I found that the bacterial flora of the various preparations, fluid and solid, varied enormously, many of them being inert with regard to milk-curdling properties, many of them containing all sorts of extraneous organisms with no useful action. In the last year or two I have found the preparation known as lacteol apparently beneficial in some cases, and at the present time I regard it as the most useful of all these products. Like the others, however, it is not in my expereince a reliable preparation.

The application of an abdominal support, carefully made and adjusted, is often of great service. The form I recommend most frequently is a spiral corset with appropriate padding.

4. *Stimulation of the Renal Excretions.*—An important point in the treatment consists in stimulating the functional activity of the kidneys. A deficiency in the total amount of water combined with excessive acidity is very commonly met with. Reference was made in an earlier paper to other manifestations of urinary disorders, more especially the presence of catarrhal cells and bacteriuria. A simple and efficient way of stimulating the kidneys consists in the administration of 12 ounces of hot water three times a day on an empty stomach, preferably in the early morning, one hour before the mid-day meal, and last thing at night. The application of the hydro-therapeutic measures referred to under skin are also indirectly beneficial in increasing the functional activity of the kidneys. In cases associated with high acidity of the urine with the presence of catarrhal cells, benefit is obtained by the administration of urotropine with acetate of potassium or other alkali.

5. *Hydro-therapeutic Measures.*—The striking effects of intestinal toxæmia on the skin are well seen in advanced cases of the disease. A general thinning of the skin with pigmentation are the most striking manifestations. These are probably indicative of long-standing disturbance of nutrition affecting the blood-vessels, sweat glands, and sebaceous glands. Whatever the explanation of the condition may be, there is no question that in the earlier stages of the disease hydro-therapeutic measures are of great value in treatment. These do not require to be very elaborate in order to produce satisfactory results. My practice consists largely in the following. I recommend a hot bath at night three times a week, to which is added two tablespoonfuls of household ammonia; the duration of the bath should be from 5 to 7 minutes, instructions being given for the skin to be thoroughly shampooed. At the same time a morning dip into perfectly cold water, or water which is as cold as possible consistent with a healthy reaction afterwards, is recommended, this to be followed by brisk friction. The use of a spray of hot followed by cold water or a needle douche is of great value when obtainable. These procedures are useful in restoring a healthier condition of the cutaneous vessels and glands, and are also useful from their invigorating effect on the whole of the nervous system. In many cases it is advisable later to recommend a Turkish bath to be taken at regular intervals, say once a week, the duration of the bath being carefully regulated, and the necessity for complete rest for an hour after it being enjoined.

6. *Massage and Exercise.*—Under modern conditions of life

many people take too little exercise. This is illustrated in the case of the young woman who is in an office all day and takes a car to or from her work, and in the case of older women in more affluent circumstances, whose exercise is largely restricted to getting in and out of a motor car. Under these conditions the whole system of internal or tissue respiration is radically defective ; an undue tax is laid upon the organs of elimination, and all the tissues suffer from a greater or lesser degree of toxæmia. In the correction of this, graduated exercise in the open air and carefully devised special exercises for home use are of great benefit. When feasible, golf and horseback exercise are of great value. When these are not obtainable, their place should be taken by some exercise in the open air combined with home physical exercises adapted from one of the systems in common use. As a matter of experience it is no use recommending patients to do *very much*, because they will give it up after a few days. It is far better to recommend one or two simple exercises to be carried out for 5 or 7 minutes night and morning, the rationale of these exercises being explained to the patient. I attach special importance to the cultivation of slow and deep inspiration and controlled expiration, in association with simple flexion and extension of the trunk muscles. Some time ago Mr Ericsen drew my attention to one exercise which has, I think, outstanding merit because of its influence on the diaphragm and on the liver. It is performed as follows : Body upright, the weight of the body supported on the extended right leg, the left leg being flexed, and the toe resting on the ground, about 8 inches behind the right heel ; the right arm is fully extended downwards parallel to the right leg, the left arm is fully extended upwards above and to the side of the head ; a deep inspiration, with free abdominal movement, is then taken, the breath is held, and the patient bends forwards to the right as far as possible, thereafter resuming the erect position, and letting out the breath at the same time. This is repeated 8 or 10 times. This exercise, carried out thoroughly for 5 minutes, is one of the best physical exercises that I know. It is useful to combine it with a similar exercise in which the weight of the body is supported on the left leg, the position of the arms being reversed. Where exercises of this kind are not feasible, their place must be taken by massage, accompanied by such active and passive movements as are possible. I attach special value to the use of vibratory massage to the abdomen with an electric motor of high speed driven with a vibratory instrument with rubber or vulcanite extremity applied to

the abdomen, beginning with an application of from 10 to 15 minutes, and gradually increased to 30 minutes once daily. I am satisfied that in many instances this has a remarkably beneficial effect in increasing the tone of the abdominal and intestinal muscles, in promoting a healthier state of the circulation in the intestinal mucous membrane and in improving the tone of the spinal and sympathetic nervous system in the abdomen, and in this way getting rid of the toxæmia.

7. *Vaccines.* In an earlier paper it was pointed out that there are cases of intestinal toxæmia in which the careful bacteriological examination of the urine reveals evidences of a true bacteriuria, the organisms I have met with being, in order of their frequency staphylococcus albus, bacillus coli, and a cocco-bacillary form of organism. In many cases the treatment above indicated has been supplemented by the use of a vaccine prepared from organisms present, with apparent great benefit to the patient. Vaccine treatment in these cases should only be regarded as supplementary to the other lines of treatment. As I have already indicated, the principles underlying the treatment of these cases is to diminish the bacterial activity at its source and at the same time endeavour to raise the natural resistance of the tissues by every known means. I am quite satisfied from close observation of many cases that the use of vaccines when carefully administered is of value in supplying the final stimulus required to enable Nature to overcome the effects of the poison. Unfortunately there is a risk of vaccine therapy falling into disrepute because of the haphazard methods of administration. Here, as elsewhere, attention to detail is important. I always give vaccines subcutaneously and rely on the amount of local and general reaction as guides to the dosage and frequency of the administration. I purify the skin thoroughly by means of sterilised water followed by spirit and ether, and I avoid the local application of strong antiseptics. More especially I deprecate the application of iodine to the skin, as the local results of the vaccine are partly obscured by its use and in other cases the local results are unfavourably influenced by the use of this irritant.

In a number of cases of rheumatoid arthritis in which the urine has been sterile and in which I have not been able to readily effect a material improvement in the state of the bowel I have used with apparent great benefit a vaccine prepared from the B. coli strain of organisms obtained from the stool. I have in my possession charts in which, prior to the use of such a vaccine, the

temperature oscillated for many weeks between 98.4° F. and 99.4° F., and, coincident with the use of such a vaccine, a normal temperature was induced and maintained. This fact, coincident with an associated improvement in the general condition of the patient, has satisfied me that such a vaccine is in some cases undoubtedly beneficial. I can recall three cases in which a very pronounced improvement in the state of the stools occurred coincidently with the use of such a vaccine, the history of the cases pointing clearly to this result being one of cause and effect.

Reference must be made to the present-day use of the commercial preparations known as phylacogens. My personal experience of these preparations is limited to a series of seven hospital cases, in which I have myself used them, and to two private patients who have used them with little success prior to their coming under my observation. In one of my hospital cases—a toxæmia of gonococcal origin—unmistakable benefit followed. In two cases slight but distinct benefit followed the treatment. The remaining cases were in no way improved or had their symptoms aggravated by the treatment. On the whole, therefore, their use is not to be generally commended; reliance should, as far as possible, be placed on obtaining an autogenous vaccine.

8. *Operative Treatment.*—There has been a striking development in recent years in regard to operative treatment of these cases. The pioneer work of Arbuthnot Lane has been followed, more particularly by American surgeons, and there is already considerable evidence in favour of the great value of operative treatment in appropriate cases. In a very instructive paper by Blake of New York on "The Value of Surgical Operations for the Relief of Neurasthenic Conditions associated with Intestinal Toxæmia" the following pronouncement is made: "Operations upon patients of this class must relieve the auto-intoxication, otherwise they will only be productive of harm. In the advanced cases, in which there is marked lengthening and dilation of the colon, partial excision or exclusion of the colon is the rational procedure; and good results may be expected. On account of the severity of the operation, the greatest care should be exercised in its application. Finally in recommending an abdominal operation to relieve neurasthenia, the following four conditions must be satisfied: (1) That there is a definite morbid or mechanical perversion of the normal condition of the viscera; (2) that it is the chief underlying cause of the neurasthenic state; (3) that the

neurasthenic condition cannot be cured without its correction, (4) that it can be corrected by a definite operative procedure of only moderate danger to life."

I entirely agree with those conditions so admirably laid down by Blake.

Two difficult questions present themselves in regard to these cases. (1) Is an operation necessary? (2) The exact nature of the operation.

With regard to the first, it may be laid down as a safe general rule that operation should not be advocated until a thorough course of medical treatment has proved unavailing. An opinion as to the advisability of operation should be based on the whole clinical features of the case, no undue importance being attached to any one particular symptom or physical sign. The X-rays are of special value in diagnosis, but here again the mere existence of an abnormal delay in the passage of the fæcal contents as revealed by the X-ray examination is no indication of the necessity for operation. Intestinal stasis may exist without toxæmia, and intestinal toxæmia may exist without stasis, and both conditions are, in the first instance, purely medical affections.

In connection with the recommendation for operative treatment I attach much importance to the determination of the existence of ileal stasis, which is usually associated with dilatation of the duodenum, and delay in the passage of food through this part of the bowel. I have satisfied myself of the essential correctness of Lane's teaching, that in the majority of instances the primary defect is in the terminal portion of the ileum, the disturbances in the large intestine and in the upper part of the small intestine being secondary to that. The case for operation may be briefly summed up as follows:—

In cases which have proved intractable to medical treatment, and where there is clear X-ray evidence of the existence of stasis, operation should be recommended.

Four courses of operative procedure are available. (1) The operation of short-circuiting ileo-sigmoidectomy in which an opening is made between the ileum and the pelvic colon. (2) A colectomy, which may be partial or complete, this being necessarily a more serious operation than short-circuiting. (3) A short-circuiting operation may be performed followed later by a colectomy. (4) In appropriate cases operative treatment may be restricted to the freeing of adhesions in the ileo-cæcal region.

the removal of the appendix, and possibly the narrowing of the cæcum and anchoring in position.

The results of Arbuthnot Lane, confirmed by other surgeons, have proved clearly the great value in appropriate cases of the operation of short-circuiting and colectomy. At the present time, however, surgeons generally have had little or no experience of these operations, and extended experience of the results in the hands of the general surgeon is necessary before any conclusions are arrived at as to the real value of these operations. We must keep in view that the operation of ileo-sigmoidostomy and colectomy may be comparatively safe and yield very satisfactory results in the hands of their brilliant author, but if undertaken lightly by the general surgeon unfavourable results are certain to follow.

In the discussion which followed the reading of this paper at the Edinburgh Medico-Chirurgical Society, it was made abundantly clear that the assistance of our surgical friends had not to any extent been sought with regard to the treatment of these cases.

The main object of my paper was to draw attention to the necessity for the close investigation of these cases by modern means and the necessity of co-operation on the part of physicians and surgeons in order to arrive at a sound conclusion as to the value of surgical treatment in the cases in question. For my own part, I am satisfied that the operations of short-circuiting and colectomy will be extensively carried out—perhaps too extensively—in the near future, and it will, I am sure, be a great advantage if the caution which surgeons have shown in regard to the acceptance of Lane's teaching is maintained at a later date when the soundness of that teaching is more generally recognised.

I will conclude by repeating what was written in my earlier paper, that the conditions which I have been considering are essentially medical conditions, and that the question of surgical treatment very largely arises because of our failure to recognise and treat these conditions properly in their early stages. Surgical treatment is directed to the results of intestinal toxæmia. The duty of the physician and practitioner is to recognise these conditions in their early stages and apply the appropriate treatment when that can be completely successful.

I have much pleasure in expressing my indebtedness to Dr Hope Fowler for the large amount of time and trouble that he has taken in connection with the study of my cases by means of the X-rays. I must also express my indebtedness to Professor Ritchie,

Superintendent of the Laboratory of the Royal College of Physicians, for the facilities afforded to me for the adequate investigation of the urine and stools referred to in my paper.

Discussion

The Vice-President said they were indebted to Dr Chalmers Watson for his very comprehensive paper, for the lucid way in which he had expounded his views, and for the beautiful X-ray photographs he had shown. A very wide field for discussion had been opened up, in fact the points raised occupied one of the most active spheres of surgery at the present time, namely the proper methods of surgical procedure in these cases. Surgeons were divided in their opinions. Sir William Arbuthnot Lane advocates ileosigmoidostomy or excision of the colon. Some others operating plicate and fix the cæcum; while others merely separate adhesions. Those were the points on which they would like to hear the views of the surgeons present.

Professor Caird.—Constipation was associated with very many ailments, and it was frequently difficult to discover the causes and to eliminate one from another. In cases of simple stenosis of the pylorus with cicatricial contraction the patients suffering from most pronounced constipation, it was pleasing to find that in addition to the cure of the local condition, the constipation vanished and with it the need for the castor-oil bottle or other aperient.

The question of the part played by various adhesions was not easily solved. It was extraordinary the number of patients operated on where one expected to find definite adhesions where there were none. In his experience it was rare to find any adhesions worthy of note or worthy of being divided. Again, in the post mortem room so-called kinks and adhesions were common, and yet the patient had never suffered any discomfort, while he should have been suffering from intestinal stasis. Pathologists and surgeons had been struck again and again by cases in which the patient had suffered from an old general peritonitis from gastric ulcer or other source, in which one could almost lift the adherent viscera out *en bloc*, the mass to see then something like a Turkish sponge and yet the alimentary canal had been performing its duty in a wonderful manner. He thought one must wait some time before speaking of the results of operation for chronic constipation and in intestinal

toxæmia. The most satisfactory case he had had was that of a servant girl; the abdomen was opened and the interior found so beautifully normal that it was closed. From that day onwards the constipation entirely disappeared. The patient was to return if she had any further symptoms; she had not done so. The mental point of view had thus also to be taken into consideration. Under certain circumstances the physician might add to his method of treatment the ancient plan of dealing with incarcerated hernia, where the patient was put to bed for a month with the foot of the bed raised and every night got a dose of castor-oil. Whether the change of position would have a good effect in cases of kinks he did not know, but probably if the changed attitude was maintained for a sufficient length of time, it would undo these kinks and so enable the intestinal contents to flow along their natural channel.

Professor Russell would have liked to have heard some of the other surgeons speak before doing so himself. Prof. Caird, in the few sentences he had spoken, had thrown an immense amount of common-sense and light upon this somewhat involved question, and he could endorse fully a good many of his remarks. In the abdomen so much depended upon the diagnosis of the factor which was troubling the patient, that it was difficult to estimate aright a record of cases given by another person. That was his difficulty in discussing this paper. He had looked over the long epitome and had failed to understand why it had been printed. Dr Chalmers Watson surely did not think that the members of the Medico-Chirurgical Society of Edinburgh did not know the present position with regard to the diagnosis and even the treatment of these various abdominal conditions. It seemed to him, after reading the epitome and listening to Dr Chalmers Watson, that they had got away from the region of intestinal toxæmia altogether, and had been plunged into the quite different one of ptosis of abdominal viscera. The question of ptosis of the abdominal viscera was, of course, a very important one, and a book had been written years ago, which he (Prof. Russell) had perused before the advent of the X-rays, in which the symptoms of ptosis of abdominal viscera were more or less fully dealt with.

With regard to the X-ray photographs, he was not prepared to accept the interpretation of many of the appearances shown; he was not prepared to accept the proposition that the great majority of the stomachs shown were normal; if they were normal stomachs, they were certainly very different from the stomach of the first

man who had a beautifully normal stomach; nearly all of them showed quite a definite degree of ptosis, and the question which had to be considered was the passage of the bismuth meal out of the stomach. The passage of a bismuth meal was a very different thing from the passage of a proteid meal, and much depended on the position in which the patient was kept after the meal had been taken. He knew, from a large experience of these abdominal conditions, that when the gastric condition was corrected, as Prof. Caird had indicated, all the phenomena which might be attributed to an intestinal toxæmia and intestinal stasis rapidly disappeared. The question even from that standpoint was a complicated one. Ptosis of the transverse colon was a condition which was exceedingly common, and one which could be diagnosed without any difficulty without the X-rays, and was a condition which might be extremely marked without any symptoms whatever.

Again, with some of the slides reference was made to splashing in certain situations—over the cæcum for instance—he did not quite know what was meant by splashing; and when the bismuth meal had become concentrated and relatively dry, he did not know how splashing could be produced. The splashing referred to in the upper part of the abdomen was, he presumed, produced over the stomach, although in the photographs the position of the splashing did not correspond to the position of the stomach; that however, might be accounted for by the upright position in which the photographs were taken. So far as he could gather, what Dr Chalmers Watson wanted was an opinion as to how often and in what instances such symptoms as he had described were to be attributed to those various ptoses. No one could hold more definitely than he (Dr Russell) did the desirability of having the intestinal tract clear, but he had seen cases in which no correction of the intestinal tract would cure so-called neurasthenia; while on the other hand some persons who were in a poor, feeble, and vigourless state of health were, of course very much benefited indeed by careful attention to diet, by diminution of the quantity of food taken, and by careful attention to the evacuation of the intestinal contents. That the symptoms were due to the absorption of bacterial toxins from the dry fæces of the colon had, he thought, been greatly exaggerated, and was certainly not in a position to be spoken about as if it were fully known. They had to deal with organisms which comparatively few people knew anything about. While recognising that neurasthenic and rheumatoid arthritic cases might be very materially benefited by

rigid attention to diet and to the efficient removal of intestinal excrement, it by no means followed that neurasthenia and rheumatoid arthritis were determined by these intestinal conditions.

With regard to kinks and perhaps even bands, he agreed with Prof. Caird. The extraordinary thing to him was that in those cases of ptosis of the transverse colon, where the hepatic flexure and the splenic flexure are practically fixed points, there was an acute bend both at the hepatic flexure and the splenic flexure, and yet in spite of that the rectum got filled with bismuth without delay.

Personally he was a sceptic about the advantages of anchoring cæcums and concerning kinks. No doubt from time to time one did meet with a case where operative procedure ought to be undertaken. At the present time he had a beautiful series of X-ray photographs from a lady who he believed ought to be short-circuited and where that course had been practically determined upon. In that case there was very great difficulty in the passage of the intestinal contents from the ascending colon, and as no measures adopted had relieved the condition, it seemed to him, short-circuiting was called for. He had, however, seen short-circuiting done, not altogether with his approval, although certainly not done against his wish, where there was general ptosis and without any relief. Short-circuiting was not to remove all the phenomena in these cases: the question was a much more difficult one than that. He denied that the colon was the part of the viscera which always ought to be attacked. In one of his cases, he thought if a gastro-enterostomy instead of short-circuiting had been done, the relief to the patient would have been infinitely greater. Before short-circuiting came into vogue, he had the condition of the stomach dealt with, and he had not become a convert to the other method.

Dr Spriggs thanked the Society for allowing him to speak, and said he rose to support very strongly Dr Chalmers Watson's plea for a careful series of X-ray examinations in cases in which intestinal toxæmia was suspected. It would, he said, only be by thorough investigation that the question as to whether " intestinal toxæmia " is an entity could be settled, and showed slides illustrative of patients sent to Duff House as cases of intestinal toxæmia in which other conditions were found.

In one case a series of X-ray photographs extending over three days showed that the condition was one of " spastic " constipation. A soft diet, leaving little residue, gave relief. The constipation

and its attendant toxic symptoms had been made worse by irritation of the descending colon by the rough food previously prescribed.

The second case was that of a man who certainly was found to have an unusually long colon, but other deviations from the normal, which were probably more closely associated with the symptoms, were discovered by urinary examination.

The third case was that of a man who had been losing weight for three years. He had had X-ray photographs taken at various times, but had never had a complete series taken after a bismuth meal. He had been operated upon for constipation, a kink in the sigmoid region having been dealt with, and appendicostomy done. His constipation was rather better, but a general feeling of discomfort in the upper part of the abdomen continued and was attributed to secondary indigestion. It was ascertained, by X-rays and by the stomach tube, that the patient did not empty the stomach till seven to nine hours after a meal. With daily lavage he began to improve, putting on 2 stones in 2½ months, and it was now clear that the source of trouble lay in the stomach.

One point about the X-ray examination was whether it is necessary to give a purge. Some people say one ought, others do not mention it. On getting his assistant Dr Gompertz, to investigate the point, it was found that the rate of passage of material was a little quicker in one case without the purge than with it, in another a little slower, and in a third there was no constant difference. It was better to prescribe a preliminary purge because one could not give an aperient during the X-ray observations and some of these patients suffered great discomfort if the bowels were not opened naturally; but if it was found inconvenient to give castor oil it could be left out without seriously affecting the results.

Sir James Mackenzie Davidson had come to hear something of intestinal toxæmia, which was the heading of the paper, but little had been said about its clinical manifestations; he had, however, been very much interested in Dr Watson's communication, and thought he had shown a wise discretion in not claiming too much in connection with his cases.

In their bearing upon the subject of intestinal toxæmia these radiographs were very interesting; but the question is, granting all this intestinal stasis, how does the toxæmia arise? Intestinal stasis must occur in multitudes of people, but not all suffer from intestinal toxæmia. There must be some natural defensive action

which prevents harm arising from the slow passage of the contents of the bowel, and it can only be when that defensive action breaks down that intestinal toxæmia can come into being. They knew that toxæmia did occur and that it might occur in various ways. He had seen a fæcal ulcer from long-continued constipation produce a diffuse abscess of the liver, and had been able to trace the connection between the ulceration and the liver abcesss. At other times there were symptoms referable to the general system. While he thought it was true that bacterial and chemical action must account for a good deal of intestinal toxæmia, there had been very little said about the condition of the intestine itself—about the intestinal wall. It was difficult to tell to what extent the canal itself may not be responsible for the poisoning of the system, not merely the bacteria but the condition of nutrition of the intestinal wall.

In connection with the surgical features of the case—kinks, adhesions, etc.—it must be remembered how nature adapts herself to difficulties within the abdomen. Sometimes one found a tumour filling the whole abdominal cavity and compressing the intestines so much that one wondered how the contents could be transmitted at all, yet the bowels acted naturally: he had seen more than one instance of that kind. He thought the physician ought to deal for a lengthened period with cases of intestinal stasis; but he agreed with Dr Chalmers Watson that in a case of manifest intestinal toxæmia, if there was reason to believe that medical treatment had been pursued without any success, the surgeon might be called in. Perhaps the success which attended surgery of the abdomen had helped to make physicians a little less patient in the investigation and treatment of the symptoms which the patient presented. One was tempted (he spoke for himself, and was sure others would agree), when confronted with a difficulty, to stop short in one's inquiry and to say, "We must have a surgeon here," when perhaps a little more patient investigation on our part might by itself have elicited the truth. The surgeon's help is invaluable, but it should not supplant efforts which are within our power, and are our duty, to make in the elucidation of a perplexing clinical problem. Moreover, in this connection it is not without advantage to consider what we should wish done were the case our own personal one.

Dr MacGillivray confessed that he had twice attended meetings of the Society on this subject with the object of getting some practical explanation of the condition, and of what was best to be done in

those cases of intestinal toxæmia associated with colitis. On the previous occasion they had had a very interesting theoretical evening, and on this an interesting evening of X-ray photographs, but he did not know that he would leave the meeting any further on as regards what was best to be done in a case of septic colitis with all the symptoms of toxæmia which were associated with that condition. He could not say that he had received any definite information or any very clear explanation of the whole condition. His personal view was that the normal alimentary canal acted as a filter which prevented any absorption of bacteria and their toxins. In the case of the back of the throat which one could see, one knew that bacteria were generally present, but no absorption; but given a chill with a local congestion and lowering of vitality and powers of resistance, and absorption with fever occurred. The same held good as regards the intestines: so long as the mucous membrane remained healthy, there might be millions of bacteria and any amount of toxins present, but no absorption. But should catarrh occur from any source of irritation then the filtering action of the wall of the bowel became impaired and consequent absorption and toxæmia occurred.

Dr Torrance Thomson said it seemed to him that they had been concentrating on the intestines and had forgotten the individual. Dr Chalmers Watson had shown a series of photographs to demonstrate intestinal stasis; he had also expressed the opinion that such a disease as rheumatoid arthritis was due to this condition of stasis. Further, he had presumed that it would be taken for granted that the lack of energy noted in the cases referred to was a result of the intestinal stasis: that was a presumption which he (Dr Thomson) hardly thought justifiable.

The criticism he would offer was that it seemed a pity that Dr Chalmers Watson had not touched upon the cause of all the phenomena associated with intestinal stasis. He had suggested kinks of various sorts as a possible cause, and as far as he understood had demonstrated their presence, but in dealing with the clinical aspects of a question of this sort, he thought one was justified in expecting that the cause of the condition would be fully gone into. He did not think that, apart from the kinks, Dr Chalmers Watson had suggested any cause for stasis.

Personally his view was very strongly in favour of looking on intestinal stasis as a local manifestation of a general condition. The danger at present was the concentration of too much attention

on this local condition with consequent neglect of the more general state. He suggested the probable psychical origin of a great many of these cases of nervous debility and lack of vigour with their resulting effects on all the muscular structures of the body, including the musculature of the intestines and abdominal parietes.

There was great risk of losing sight of the " fons et origo " of the whole condition, which he believed to be some constitutional change.

Mr George Chiene asked whether the fæces examined were acid or alkaline. Dr Chalmers Watson would find pin-head oatmeal a very excellent method of treating the non-surgical cases. As regards surgical treatment, he thought anchoring the cæcum should be given up: to anchor the bowel to the abdominal wall was, in his opinion, undesirable. The question whether one ought to deal with the condition in the region of the cæcum or by Lane's method had arisen, and the advantages and disadvantages of the two plans set forth. In connection with the latter there were two points: the first was, that in a certain proportion of cases diarrhœa was a prevalent result, lasting from a short time after the operation up to a much longer period; the second and more serious point was regurgitation of the intestinal contents, which are poured into the sigmoid, backwards into the descending colon. That was probably the most serious after-condition which occurred. Personally he could not speak of anastomosis in connection with intestinal stasis, but he had experience of it in cases of tuberculous disease of the ileo-cæcal valve. Ten years ago he had shown a child in whom an anastomosis had been performed between the ileum and the transverse colon, and since then he had performed the same operation in similar tuberculous cases with good results. Mr Struthers had recently shown a case treated in a similar manner. In those cases there was no question of either backward regurgitation or intense diarrhœa. Mayo in 1911 suggested a method of anastomosis into the transverse colon, plus the removal of the cæcum. If Dr Chalmers Watson would try the operation he suggested, he would find that he gets the advantages of Lane's method without the disadvantages.

Mr Dowden wondered if Dr Chalmers Watson had read a paper by Rosving of Copenhagen, on the causation of visceroptosis.

Rosving pointed out that the majority of these cases occurred

in women, and generally in young women. The normal figure was altered, and this alteration was due to interference with the normal development of the lower ribs through the action of corsets, not unduly tight. As a consequence, the large right lobe of the liver was pressed down, and the kidney became more mobile and palpable, and this naturally accounted for its almost invariable presence on the right side. All the other abdominal viscera were consequently in a state of ptosis.

Mr Dowden had performed short-circuiting in apparently suitable cases without satisfactory results. Gastropexy he had also done after Rosving's method on three occasions, and only one was a cure.

Mr Struthers referred to the difficulty in interpreting the appearances presented in Dr Chalmers Watson's radiographs owing to the fact that our knowledge of the behaviour of bismuth meals in normal subjects was not yet complete, and he suggested that it would be of great value if some one would make a complete series of observations on the normal and present them for the consideration of the society. As an instance of the varied interpretation possible he referred to the statement made that a low position of the transverse colon, as shown, was abnormal. It was well known to anatomists and surgeons that the position of the transverse colon was subject to great variation, and a low position of that part of the intestine was, *per se*, probably of no clinical significance whatever. Like others who had frequent opportunities of opening the abdomen Mr Struthers had taken an interest in the various theories advanced to explain the origin of Jackson's membrane and other so-called adhesions to which significance was attached by some. From what he had seen he had come to the conclusion that they were, in the main, anatomical in origin, i.e. they were due to variation in the manner in which the intestine became finally attached to the abdominal wall when growth was complete, as suggested by Gray and Anderson of Aberdeen and others. Granting that this explanation was correct, had they any significance? Mr Struthers instanced a case in which obstruction had been produced by Jackson's membrane, but stated that as a rule the adhesions found were not such as to interfere with peristalsis. In discussing the operation of short-circuiting, Mr Struthers referred to three cases in which he had done this operation, in two of them for chronic tubercular disease of bone and in the third after resection of small intestine for strangulation. In the two done for tuberculosis no benefit resulted. In the third the

cæcum and ascending colon filled up with dry solid fæcal material and had subsequently to be resected. The fact that the cæcum and a large part of the colon were apt to fill up in this way after short-circuiting seemed to him such a serious drawback that the future for the operation was not a very hopeful one.

Mr E. Scott Carmichael joined with Mr Struthers with regard to the interpretation of the X-ray photographs ; the more one studied these cases, one had to lay less and less stress on the anatomical position of the abdominal viscera ; and he was not sure but that one must come to the same conclusion as to the time a bismuth meal takes to pass out of the stomach. This must vary in different individuals and at different times in the same individual. He had taken a particular interest in these cases as well as others before operation, and had in many cases examined them by means of the screen as well as the photograph. The photographs did not always give an accurate picture, and when one had seen the actual movements of the stomach and intestines, both small and large, by means of the screen, one was in a better position to interpret. Surgeons did not see screens often enough. The difficulty they had was in accepting the diagnosis of the radiographer ; the radiographer did not come to the operation and see what was found after making a diagnosis. Sometimes, he thought, the radiographers were too dogmatic in their diagnoses.

As to bands, he quite agreed with Mr Struthers that the more one opened the abdomen, particularly in women, one saw all varieties of kinks and bands in patients without any intestinal symptoms.

Then with regard to operation. Many of the cases of so-called chronic appendicitis are really cases of cæcal colitis. In those cases the question arose whether any operation should be performed at all or whether the cæcum and colon should be removed. Personally he felt that tacking down the cæcum and colon was an unsurgical and unsatisfactory operation. If operation was determined upon, it was better to remove the cæcum and ascending colon, and perform anastomosis between the ileum and transverse colon.

Mr Henry Wade said he gathered from the physicians who had spoken that a class of case existed which, perhaps for the lack of a better description, was at present described as one of chronic intestinal stasis, and that such cases not infrequently ultimately

required surgical treatment for their relief. He entered very strongly, however, his dissent from the opinion expressed by Professor Russell, who, altogether condemning strongly the form of surgical treatment recommended by Dr Chalmers Watson, at the same time recommended another, which was the performance of a gastro-enterostomy for the gastric dilatation that was met with in these cases. The speaker had been trained to consider that the performance of such an operation, except for stenosis of the gastric outlet, or an actual ulcer of the stomach or duodenum, was unjustified except under the most exceptional circumstances. His personal experience also bore this out in that, where gastro-enterostomy was performed for an atonic gastric dilatation, the results were harmful to the patient. The reason why the speaker in common with certain other surgeons, hesitated to perform the operation of short-circuiting for the treatment of intestinal stasis was the liability of regurgitation to occur into the cul-de-sac of the colon and cæcum above the anastomosis. This complication was not the result of faulty technique, for it appeared that even in the opinion of Sir Arbuthnot Lane, a definite percentage of cases, where a short-circuiting operation had been performed subsequently required to have a total colectomy performed, owing to regurgitation having produced fæcal accumulation in the cul-de-sac.

The ideal treatment for intestinal stasis was undoubtedly the operation of total colectomy. Prior to having seen this operation performed a few days ago by Sir Arbuthnot Lane, the speaker had looked upon it as a terrible and dangerous operation. As carried out by Lane, however, it was an operation of great beauty and no very grave severity, so much so, that the patient, who before the operation had a pulse rate of 76, had that evening five hours after the operation, a pulse rate of 80 per minute. The speaker was not however, prepared to suggest that a similar success and safety would follow its performance by less brilliant surgeons.

A previous speaker had referred to the operation of cæcal plication combined with cæco-pexy as now original. With this contention he did not agree, and would desire to examine the grounds on which such an operation was undertaken.

In order to understand the rationale underlying such treatment as partial colectomy or cæcum plication and cæco-pexy, it is necessary to remember the physiology of the human cæcum and the deformities frequently met with in it.

Throughout the alimentary canal, two cavities exist within

which the food is retained for some considerable time in order to undergo digestion. The first is the stomach, where chemical digestion takes place, and the other is the cæcum or stomach of the lower bowel, where the putrefactive or fermentative digestion of cellulose should naturally occur.

The analogy between the stomach and the cæcum has been shown to be extremely close. On anatomical grounds, Keith has demonstrated the natural sphincter guarding its outlet, the cæco-colic sphincter. Wilkie has pointed out how this region, like the pylorus of the stomach, is frequently the site of inflammatory or malignant disease. Canon, working at the experimental physiology of this region, demonstrated that, within three and a half hours of ingestion, the waste products of digestion that have lost 97 per cent. of their nutritive value are passed through the ileo-cæcal valve into the cæcum. From this situation they are carried by a regurgitant wave into the fundus of the cæcum, and here accumulate. During this period, the cæco-colic sphincter remains closed and only allows small quantities of the cæcal content to escape at regular intervals into the colon beyond. During its stay within the cæcum, food debris is acted upon by the bacteria within, and the extent of this secondary digestive process will depend upon the nature of the food material. In a coarse diet rich in cellulose it is of course a valuable process. In modern man, however, it is of little value from the point of view of providing nutriment. It permits, however, of absorption of a large quantity of the fluid element of the intestinal content.

A point of the utmost importance naturally arises, when we consider what is the natural stimulus to the further progress of the cæcal content. This does not appear to be analogous to that found in the stomach, where, after digestion has reached a certain stage, the chemical alteration of the intragastric content excites a nerve stimulus that produces a pyloric relaxation so that the acid chyme is jetted through into the duodenum. The stimulus would appear to be similar to that which regulates the passage of the intestinal content along the bowel, and produces peristalsis. This stimulus is not chemical but mechanical, increased pressure at one point within the bowel causing a contraction of the bowel above and relaxation of the bowel below, this being known as "the law of the intestine," and is observed as peristalsis. If this contention be correct, it follows in consequence that two factors are of great importance in determining the rapidity with which the content of the cæcum is expelled ; the first being the bulk of the food débris

entering it, the second being the size of the cavity. Thus pronounced natural cæcal stasis would appear to owe its origin either to food that is too perfectly nutritious so that its bulk is reduced to a small amount when it enters the cæcum, or to a cæcum which is unduly distended. The latter class of case is usually associated with what is termed "Cæcum Mobile."

It is in those cases of cæcum mobile with associated intestinal stasis that the operation of cæcal plication and cæco-pexy is especially suitable. Frequently in such cases the cæcum is found occupying the pelvis and, when withdrawn from it, it comes out with a suction-like action. The plication of the cæcum not only reduces its cubic capacity by over a third, but, at the same time, lifts it up into its natural position in the false pelvis. If the cæcal stasis in such cases is the result of cæcal dilatation and consequent retention, it would appear that the most satisfactory treatment would be the performance of an operation such as has been described, which, by reducing the dilatation, expedites the expulsion of cæcal content.

Mr Wilkie thought that the time had come for them to take up some definite attitude with regard to the subject of intestinal stasis. They could not afford to ignore the results achieved by Lane by the operations of short-circuiting and colectomy. This subject was commanding widespread interest among surgeons, but as such far-reaching issues were involved it deserved to be investigated much more thoroughly before such operations came into general use. One would like to have some definite scientific evidence as to what was being absorbed and causing these secondary diseases. One was a little disappointed to find in Lane's clinic that more of a scientific nature was not being done in conjunction with his operative work. He (Mr Wilkie) was firmly convinced that trouble in the colon and in the appendix played an important part in the etiology of duodenal ulcer. He granted that at operation it was very difficult to locate such trouble through an epigastric incision, as it was frequently impossible to obtain a perspective view of the abdomen. In quite 75 per cent. of cases of duodenal ulcer examined post mortem there was found clear anatomical evidence of stasis in the colon.

The immediate results of conservative operative measures were often extremely satisfactory. He had found that plication of the cæcal wall without fixation of the cæcum gave good results in those cases with a large prolapsed atonic cæcum, which were so frequently confused with cases of chronic appendicitis. In two cases sent to

hospital with the diagnosis of appendicitis and general peritonitis he had found on opening the abdomen no trace of inflammatory trouble, but in each an acutely dilated and prolapsed cæcum which had become wedged in the true pelvis. Plication of the cæcum was followed by complete relief of symptoms in both cases. The reduction in the length as well as in the calibre of the cæcum after this operation was very striking, and he could not agree with those who regarded it as being unsurgical.

Dr Chalmers Watson in reply thanked the Society for their appreciative hearing of his paper. His paper was to appear *in extenso* in the "Transactions," and some of the points raised by the speakers would there be dealt with. Much of the criticism from Dr Russell and others was of great interest but not relevant to his demonstration. He could not agree with many of the views expressed by Dr Russell. Dr Russell stated that the splenic and hepatic flexures were anatomically fixed points. The information given by the X-rays had long ago shown this to be a fallacy.

The main points that the speaker had raised were the frequency with which cases that he had described were met with by his colleagues and other practitioners, and the inefficacy of medical means in their hands to relieve the condition, and of the desirability of having recourse to operative treatment in cases where medical means failed to relieve. That was really the crucial point in which the speaker had desired to hear the opinion of the meeting.

In his demonstration the speaker referred in detail to many cases in which the results of his medical treatment had failed to give a satisfactory result. He found that in other parts of the world surgeons were being invited to do something for the relief of these patients by short-circuiting, or other operation, and were doing so with reported large measures of success, and he wished to ask his medical colleagues whether the time was not ripe for the investigation of these cases from this standpoint. Were they content to leave their patients unrelieved when their surgical friends elsewhere were doing something for them? He did not think they should take up such a position, but advocated a preliminary investigation of the cases, utilising to a much greater extent than had been done all the modern methods of investigation, including the X-rays. The X-rays were valuable in these cases, but were of no use unless a systematic series of photographs were taken, the results being considered along with the other clinical findings.

The whole object of his paper was to try and arouse interest in a subject which had not yet here received the attention it deserved. Some of the surgeons had expressed an adverse view as to the prospects of success of operative treatment, this view, however, not being based on practical experience.

In concluding, Dr Chalmers Watson advocated a closer co-operation of the physician and surgeon in the investigation and treatment of the cases.

Meeting V.—February 1914

Dr Melville Dunlop, *Vice-President in the Chair*

I. Election of Members

Walter Waddell Carlow, M.B., Ch.B., F.R.C.S.Ed., 20 Eglinton Crescent; William Hope Fowler, M.B., Ch.B., F.R.C.S.Ed., 21 Walker Street, were elected Ordinary Members of the Society.

II. Exhibition of Patients

Dr Byrom Bramwell showed—

Stone-mason suffering from diffuse scleroderma.

Dr George Mackay showed—

(*a*) A case of congenital cataract in a child of fourteen months, illustrating unusual rapidity of absorption of lens matter after needling. The case further illustrates tuberculosis in a bottle-fed infant, and right facial paralysis after operation on the mastoid and cervical glands.

(*b*) A case illustrative of trephining for glaucoma combined with peripheral iridectomy.

III. Exhibition of Specimens

Dr Chalmers Watson showed—

(*a*) Ascaris lumbricoides.

(*b*) Ova of Ascaris lumbricoides and ova of Trichocephalus dispar from fæces of same patient.[1]

[1] Lady home from India suffering from anæmia with intestinal disturbance, diagnosis being made by passage of worm when patient taking hot bath.

IV. Original Communications

1. ON THE RÖNTGEN RAY AND BISMUTH AS AN AID IN THE DIAGNOSIS OF SOME ALIMENTARY DISEASES

By Edmund Price, M.B., C.M., Radiologist, Chalmers Hospital, Edinburgh

Mr Chairman and Gentlemen,—When, at the beginning of the Session, I offered to give this demonstration, it had been my intention to bring before your notice the information gained by the radiographic examination of the whole alimentary tract, but on going over the material on hand it became evident that in so doing I ran the risk either of boring you to distraction and crowding out other equally interesting communications, or of doing my subject scant justice, and so with your permission I shall to-night omit all reference to diseases of the stomach, leaving this for some future occasion, and confine myself to the consideration of some of the conditions in the large intestine in which radiography proves useful.

I should be wanting in a sense of proportion if I did not at the very outset remind you that the X-ray and opaque meal is only one of many means that we are familiar with for arriving at a correct diagnosis in alimentary diseases. Indeed I think its most brilliant achievements lie rather in the direction of confirming conclusions already arrived at by the other and better-known method than in that of making independent discoveries, and that the nearer we approach to perfection with skiagrams, the greater is the necessity for a thorough clinical examination. But whilst this is my belief I must also remind you that there are few cases in which radiography cannot give you some help, and that in cases of intestinal stasis and allied conditions its aid is indispensable. We cannot forget that, but for the work of Alfred C. Jordan, Arbuthnot Lane could not have opened up the new field of surgery with which his name is associated, and that nearly everything worth knowing about the normal intestine has been taught us by another great radiographer, Hertz.

Gentlemen, a remark was once passed that more mistakes had been made in diagnosis by the X-ray method than by any other, and I must admit that there was a considerable amount of truth underlying it, the reason being that we were unfamiliar with the normal radiographical appearances of the alimentary tract. The writing was all Greek to us because we did not know the alphabet. In the second place we were apt to ignore, to pass over as of no account,

clinical facts discovered by other methods, and to depend entirely on the radiogram, which were, and I am speaking only of my own work, poor at the best; and lastly we were not alive to the importance of examining the whole alimentary tract. When we remember how closely interdependent the organs of this system are we can realise how much useful information we must have missed when we confined our observation to the stomach alone or to the large intestine alone.

As regards technique, I always endeavoured to be allowed to make a complete examination, and, remembering the remark frequently made by, amongst others, Prof. Russell, that the bismuth meal is one thing and the ordinary meal another, I endeavour to make the meal as palatable as possible, and have found that bread and milk have proved most acceptable to my patients. Ninety per cent. of them say it is very good indeed, five per cent. say it is better than they expected it to be, and the remaining five—well, I don't believe they would be pleased though I were to bring down manna from heaven. The entire meal consists of 2½ ounces of bismuth oxychloride, or Mercks' barium sulphate, 2½ ounces of bread-crumbs, about 6 ounces of hot milk, and a little sugar; the whole is carefully mixed and served as daintily as possible. During the time the patient is taking the meal I carry on a conversation which I make as diverting as my mother wit will allow of my doing. After the meal is finished the patient undresses, the time thus occupied allowing the meal to settle in the stomach and separate itself from the gases always present. I then place an opaque disc on the umbilicus and make my first screen examination, centring the tube over the anatomical position of the pylorus. After having made a careful screen examination I expose a plate. All my examinations are made with my patient in an upright position. In order that there may be no risk of the bowels moving during the period under which they are under examination, I advise that these be thoroughly emptied the day before the first examination is made; and in order that I may interfere as little as possible with the patients' daily routine, insist upon them coming to me about 9 o'clock in the morning, without having any breakfast. The I supply in the form of the opaque meal. The next examination is made at 1 o'clock, the patient receiving strict injunctions to take no food, solid or liquid, in the interval. After the second examination the patient is allowed to take lunch, afternoon tea, dinner as usual. No purgative is administered during the time the examination is being under-

gone. Taking Hertz' time-tables as my guide, I find that making examinations at intervals of $3\frac{1}{2}$, 8, 12, and 24 hours, after the first one, give all the information obtainable in most cases, though one has occasionally to go on for 36, 48 hours, and even longer. Very often the 12-hour examination can be omitted, this depending on the rate of progress observed on the screen. Finally, gentlemen, in the series of skiagrams which I show you to-night I have endeavoured to demonstrate some of the directions in which radiography may be of use to you, and beginning from a series taken from a normal individual, have tried to go gradually down the scale to those cases of advanced intestinal stasis in which an operation of some description depending on the discretion of the surgeon is urgently called for. These skiagrams will, I believe, convince you that by means of radiography we can demonstrate the exact *position* of the various organs comprising the alimentary system in health and also their variation in disease. Further, we can give you accurate information regarding their *conformation* under the two conditions, though you must always bear in mind that it is the shadow and not the reality that you are observing. Thirdly, you can learn something about the *rate of progress* through the alimentary canal of its contents, and should delay be one of the conditions present, your attention will be directed to the affected area with unerring truth; and lastly, it can give you invaluable information with regard to the *condition of the walls* of the various organs, whether they are healthy or in a state of degeneration and atony. So that in those cases in which an operation is called for, the surgeon has at his command a method of investigation which enables him to decide before opening the abdomen the nature of the operation he will probably be called upon to perform, and the extent of diseased intestine he has to deal with. And this point is well illustrated, I think, in case 4, in which there is clear radiographic evidence of degeneration in the cæcum, ascending colon, hepathic flexure, and right half of the transverse colon, and in which Mr Stiles, instead of doing a complete Lane's operation, resected the portion of large intestine mentioned, and planted the ileum into the healthy left half of the transversal colon with very happy results, as far as the patient is concerned. Case 5 shows an even more advanced stage of atony, involving as it does the whole of the colon. Prints of this case were shown to Sir Arbuthnot Lane, and on the strength of them, and of course of all the other clinical evidence, he advised that a colectomy be performed. Case 6 illustrates another direction in which the

radiographic method may prove valuable. It is that of a woman, 41 years of age, with well-marked symptoms, usually attributed to intestinal stasis, headache, malaise, constipation, and unfitness for work. She measured her constipation by saying that she needed to take an infusion of a handful of senna pods every night in order to get her bowels moved. You see from the slide that she has a very low colon, and there was marked delay in the cæcum and transverse colon. Mr Stiles performed an ileopelvicolostomy, and she came back eight months afterwards to report herself. She was now perfectly well, and able for all her duties. The headaches had entirely disappeared, and all that was left of the constipation was that she occasionally, once a week or so, required to take 4 or 5 senna pods. In order to see how the short-circuiting operation was doing, I took this radiograph 12 hours after administering a bismuth meal, and observe what has happened. A great part of the food has regurgitated into the transverse colon, and presumably some of it lies there until such time as she has taken her 4 or 5 senna pods. And yet all the symptoms that are supposed to be due to absorption from the large intestine have disappeared. One swallow does not make summer, but in this case at any rate I think we can say that whilst the operation of ileopelvicolostomy, by giving freer egress to the contents of the ileum, has completely relieved the patient of all her distressing symptoms, it has not removed one of the supposed causes of the symptoms, namely, faecal accumulation in the large intestine and the subsequent toxic absorption, and the question arises, Does absorption take place through the atonic atrophied walls of the large intestine? This case at all events seems to beat out Prof. Adami's contention that absorption does not take place, at any rate to the extent it is supposed to do by some physicians.

Discussion

The Vice-President (Dr Melville Dunlop) felt that the Society was very greatly indebted to Dr Price for his very precise and delightfully clear paper, also for his altogether beautiful demonstration. Edinburgh was very fortunate in having a radiographer of such eminence as Dr Price, who not only produced most beautiful radiographs, but was always willing from his experience to interpret their meaning to others. The subject was interesting both to the physician and surgeon, because those abdominal conditions were often exceedingly difficult to diagnose, and it was only by means of

these radiographs that valuable assistance could be brought to bear upon the subject.

Dr Burn Murdoch expressed his thanks to Dr Price for his able paper, and thought that the Society owed a debt of gratitude to him for the excellent demonstration they had just witnessed.

He emphasised what Dr Price had so clearly pointed out, the use of radiography in studying difficult cases. Personally he had found it enable him to come to a more definite diagnosis in obscure abdominal cases. Dr Price had referred to one of his patients, where obscure symptoms which had persisted from early childhood had been cleared up by the use of radiography. It afforded evidence of aggravated retardation at the hepatic flexure, which fortunately yielded to treatment by liquid paraffin. In pre-radiographic days this patient had been treated by very able men unsuccessfully. Dr Burn Murdoch emphasised the fact that it was the radiograph which enabled him to get at the root of the matter.

In many other cases of difficulty, where the question of operation arose, he had also found Dr Price's radiographs of great help. In those difficult cases of neurasthenia, accompanied by displacement of the abdominal organs, in which dropped kidney was almost always present, he had also derived help from radiographs. He instanced one case in particular where, had it not been for the information afforded by X-ray photography, an operation would probably have been undertaken. This patient was under five stone in weight, but under Weir-Mitchell massage treatment recovered, gaining almost three stone.

Mr Scot Skirving expressed his appreciation of Dr Price's work; more excellent skiagrams he had seldom seen. To those who knew the amount of care Dr Price took in his technique, however, it was no surprise that his results were so good. He (Mr Skirving) had got great help from Dr Price on several occasions. He hoped, however, that Dr Price would not object to a little criticism. He thought the latter was inclined to go too far, and claim too much for the radiographic specialist. Dr Price had said that in very many cases the radiographic specialist could tell the surgeon exactly what he was going to find inside the abdomen. No doubt that was quite true in many cases. Did Dr Price, however, really mean to say that, from the *inspection of the X-ray plate alone*, he could diagnose pathological thinning of the bowel wall or spasm of the

bowel, as in two of the plates just thrown on the screen, in which his diagnosis had been confirmed at the operation?

How, for example, did he in the first case (demonstration on the blackboard) exclude localised gaseous distension of the intestine as the interpretation of the skiagram? Again, take the second case of the lady whose skiagram showed the transverse colon in the form of an acute V, in which the cæcum, ascending colon, and proximal limb of the V were shown much distended with the bismuth meal, and in which the distal limb could only just be made out (demonstration on the board). How did Dr Price diagnose that the distal limb was in a state of spasm?

Surely the most natural interpretation would have been to suspect adhesions at the apex of the V (where the bismuth meal stopped abruptly), causing a kink or angulation of the bowel there?

Mr Skirving hoped these questions would be satisfactorily answered.

Dr Gulland was only too glad to acknowledge a debt of gratitude to Dr Price, and as regards his main contentions he certainly had nothing to say: he had been a help to him. Mr Scot Skirving had raised the point of spasm; he (Dr Gulland) had had a very interesting illustration in a hospital patient during the last few days. The patient was a man sent in with stomach symptoms—constipation and all sorts of things—for which no satisfactory explanation could be got. A test meal showed nothing much out of the way, and nothing very important was made out in the abdomen at all. The patient was X-rayed in the usual fashion, as all of his stomach cases were. The stomach was found to come to itself very normally, and nothing very much was seen. The passage from the small intestine to the ascending colon was all right, but at the splenic flexure there was a great collection of material (demonstrated). The photograph was a very good one indeed. The radiographer read that as a stricture at that spot. Mr Miles was asked to see the patient, and agreed that it should be investigated. Dr Gulland, however, had his doubts as to whether a stricture really existed. At the operation nothing was found wrong, and that the condition was one of spasm. The real explanation was a very tight pylorus, so tight that the tip of the little finger would not go through it. That illustrated one of the real difficulties about the interpretation of X-ray photographs, and, in dealing with mobile organs like the stomach and bowel, it was necessary to screen

these cases for a long time, otherwise one might occasionally be misled.

Mr Stiles regretted that he had not been in time to hear Dr Price's paper. He (Mr Stiles) was in the habit of making extensive use of skiagraphy in his gastro-intestinal cases, so that he had discussed the subject with Dr Price on many occasions, and he had found Dr Price's work most helpful not only in determining the nature and situation of the mischief in the conditions of atony and stasis met with in the gastro-intestinal canal, but also in deciding as to the nature and extent of the surgical procedure best calculated to combat the condition.

While the ordinary clinical examination was often sufficient to enable the surgeon to decide as to proper treatment, it was always well to have one's observations and opinions confirmed by skiagraphy. For example, he (Mr Stiles) had often found that the X-ray plates enabled him to come to a definite decision as to whether, in a case of intestinal stasis, it was advisable to resect the ileo-cæcal region along with the ascending colon and hepatic flexure, and to unite the ileum to the transverse colon, or merely to short-circuit the large intestine by performing an ileo-sigmoidostomy. In the majority of cases it was the condition of the cæcum, ascending colon, and hepatic flexure which was responsible for the stasis, and in these cases the proper treatment was to resect these parts and unite the ileum to the transverse colon. When on the other hand the whole of the large intestine was at fault, ileo-sigmoidostomy should be done in the first instance, and, if necessary, the large intestine could be safely excised by a subsequent operation.

He (Mr Stiles) had recently seen a case in which the patient was advised to have the cæcum and ascending colon excised, but X-ray photographs showed that it was the pelvic colon that was alone responsible for the stasis. Not infrequently patients suffering from an atonic and prolapsed cæcum are sent to the surgeon for appendectomy, under the belief that the appendix was the cause of the trouble. If there is any doubt about the condition of the cæcum, an X-ray photograph will generally decide the question, and will enable the surgeon to explain to the patient that something more than mere removal of the appendix may be required.

The correct interpretation of skiagrams was a matter not only of anatomical knowledge but also of clinical experience. Mr Stiles did not for one moment wish to infer that the physician and

radiologist did not possess the necessary anatomical knowledge, but he thought that the surgeon had the advantage of always comparing what he saw in the photograph with what he found when he opened the abdomen. He (Mr Stiles) was glad to notice that the physicians of the present day were beginning to realise the importance of following their cases into the operating-room. Dr Price had very wisely made it a rule to do so whenever possible.

Dr William Russell wished to add his tribute to those already paid, and to express again the pleasure it was to see this demonstration, and to hear the way in which it was made to the Society. There was really not much to say about a communication of this sort: it was a field with which they were all now more or less familiar. They were also familiar with the excellent work which Dr Price was doing in this direction. They all availed themselves of the opportunities of examining certain patients by this method. There was no doubt from time to time a little difference of opinion between the clinician and radiographer—an appendix was sometimes supposed to be where there was no appendix, and so on. Dr Price might have covered a wider field than he had done; even his (Dr Russell's) knowledge of the work that Dr Price had done covered a much wider field than he had shown that night. He was sure that not only he but all the other members of the Society, who were interested in good work of this kind, would be delighted if Dr Price would still further extend his communication.

Dr Gulland's case reminded him (Dr Russell) of a case with similar appearances at the splenic flexure, not due to spasm but to malignant stricture.

Dr Price, in reply, said with regard to Mr Scot Skirving's question about the appearance of the hepatic flexure, that when the cæcum was beginning to fill one might have this appearance in the highest part of this region of the bowel, but if after 8, 12, or 24 hours one found that the cæcum was full, and that the bismuth was going past the hepatic flexure into the transverse colon, and still there was the appearance of thin wall, they might conclude, as Mr Stiles found at the operation, and the radiograph showed, that there was a thin wall. He did not for a moment say that if there at the beginning it was an atonic wall. After the parts are filled or ought to be filled then there must be some reason for this appearance of flatulent distension, and they had proved at the operation that this was due to an atonic, thin-walled cæcum and colon.

In connection with the question of *spasm* of the colon demonstrated in the case in which the right half of the transverse colon was enormously distended with bismuth, whilst the left half was practically empty and formed an acute angle with the right half, he should have said that they were alive to the possibility of the presence of adhesions at this point, and an emulsion of bismuth was injected into the large bowel; and, on taking a radiogram with the patient lying down, it was found that the contents of the pelvic colon had regurgitated into the transverse colon, past the sharp angle, and that there was no permanent obstruction at that point. Further, they knew from the medical man in charge of the case, that the bowels moved naturally after a dose of morphia, which fact tended to confirm the radiographic finding of spasm in the left half of the transverse colon.

Mr Scot Skirving said it was not then the radiographic evidence that made the diagnosis.

Dr Price replied that they never ignored other evidence. They had the radiographic evidence when the patient was lying down, and the food given per rectum passed right down and filled the transverse colon from the hepatic flexure to the splenic flexure. If there had been a marked stricture, the food would have stuck at that point (demonstrated).

Mr Scot Skirving said that what he wanted to get at was simply this, supposing one knew nothing about the history of the patient and one took a skiagraph, could one by looking at the skiagraph in certain cases diagnose (*a*) spasm, or (*b*) a thin-walled intestine?

Dr Price replied that he would not undertake to make a diagnosis under these circumstances. If he took the X-ray himself he would be able to show the part of the bowel which was affected—he had shown two such cases, and could show several more. He concluded by thanking the Chairman for his kindness.

2. DIFFUSE SCLERODERMIA: ITS FREQUENCY; ITS OCCURRENCE IN STONE-MASONS; ITS TREATMENT BY FIBROLYSIN—ELEVATIONS OF TEMPERATURE DUE TO FIBROLYSIN INJECTIONS

By Byrom Bramwell, M.D., F.R.C.P., LL.D.,
Physician to Chalmers Hospital; Consulting Physician, Royal Infirmary, Edinburgh

Diffuse sclerodermia is a very rare condition. Dr Phineas S. Abraham, the writer of the article on "Sclerodermia," in Allbutt and Rolleston's *System of Medicine*, in speaking of diffuse sclerodermia, states that M'Call Anderson had 2 cases in 11,000 diseases of the skin, and Crocker 2 in 10,000 cases. In 17,000 general medical cases seen in my hospital practice (8502 in-patients, 8638 out-patients) there were 7 cases of diffuse sclerodermia, and in 10,000 general medical cases seen in private practice, of which I have kept notes, there were 2 cases of diffuse sclerodermia.

The first point I wish to make is that, so far as these figures show, cases of diffuse sclerodermia come at least as frequently under the care of the general physician as under the care of the dermatologist—in 22,000 cases of diseases of the skin seen by M'Call Anderson and Crocker there were 4 cases of diffuse sclerodermia, while in my 27,000 cases of general medical disease there were 9 cases of diffuse sclerodermia.

The next point which I wish to make is the relatively frequent occurrence of diffuse sclerodermia in stone-masons. In my 9 cases of diffuse sclerodermia 5 were stone-masons and 1 was a coppersmith, who was accustomed to work with a cold chisel. The only apparent cause which I am able to suggest for the relatively frequent occurrence of diffuse sclerodermia in stone-masons is the holding of a chisel in the hand during cold weather. In all of these six cases (5 stone-masons and 1 coppersmith) the condition commenced in the hands. None of the patients had suffered from syphilis so far as could be ascertained.

In his article on "Sclerodermia" in Allbutt and Rolleston's *System of Medicine*, Dr Phineas S. Abraham makes no mention of the occurrence of the condition in stone-masons. He informs me that he knows of no writer who does so. I feel certain, however, that my own experience on this point cannot be peculiar. Perhaps some general physician who reads this article will be able to corroborate my experience. I have been so impressed with the occurrence

of diffuse sclerodermia in stone-masons that in the last three cases which have come under my observation I have correctly suggested the occupation of the patient as soon as I saw and felt the hands and *before I had asked him a single question.*

The next point to which I wish to refer is the marked improvement which resulted in some of my cases of diffuse sclerodermia from subcutaneous injections of fibrolysin.

In two of the cases the injections, which at first produced no disturbance of temperature and pulse, produced marked elevations of temperature. In both cases these temperature alterations were identical in character. I have employed fibrolysin in several other diseases. In a case of disseminated sclerosis a very high temperature (105.8° F.) followed an injection of fibrolysin. So far as I am aware, no one has previously directed attention to this point.

The last point which I have to note is the close resemblance in some of the clinical features of diffuse sclerodermia to Raynaud's disease. In almost all of the cases exposure to cold produced the same cold, blue, dead condition of the hands which is such a striking feature of Raynaud's disease. In four of the cases painless sores, which were very slow to heal, developed on the knuckles or finger-tips, apparently as the result of slight injuries. In cases v. and vi. the tip of one of the fingers became gangrenous.

In one case (case v. which was shown to the Society, the condition when I first saw it $14\frac{1}{2}$ years ago was typically that of sclerodermia—the hands, face, feet, and thorax were affected. The hard, brawny, swollen condition of the hands and the hide-bound condition of the face and front of the chest have now in great part disappeared. The fingers have become bent in to the palm, and the tips of several of them distorted as the result of trophic lesions, highly suggestive of Raynaud's disease. Further, bright red vascular spots, highly suggestive of xerodermia pigmentosa, have developed on the face, tongue, chest, back, and dorsum of the right hand. This is a very remarkable case.

Hæmatinuria was carefully inquired for but was not observed in any of the cases. In several of the cases pains, apparently rheumatic, and stiffness of the joints were complained of.

The thyroid was not enlarged in any of the cases.

The clinical features of diffuse sclerodermia are to my mind very suggestive of a trophic or vasomotor neurosis, but in none of the cases were any definite objective symptoms suggestive of syringomyelia or other definite nervous lesion detected.

The clinical features of the 9 cases which have come under my observation are detailed in the following table and notes :—

Case I.—Miss F., aged 65, seen in consultation on 28th December 1892.

Complaints.—Pain and stiffness in hands and legs ; inability to walk ; shooting pains in hands and fingers, and burning sensation in the feet.

Duration.—Three years ; commenced with a bulla resembling pemphigus over the outer side of one leg.

Condition when Seen.—Right hand stiff and swollen ; skin of hands, especially palms, very hard and dense ; movement of fingers impaired. Left hand affected in same way, but in a less degree than right. Legs bent and rigid ; knee-jerks cannot be elicited.

The condition appears to be a combination of scleroderma and peripheral neuritis.

Treatment.—All treatment has failed. Sulphonal gives sleep.

Subsequent Progress.—Patient died five years later from cystitis.

Case II.—D. K., aged 30, stone-mason, seen as an out-patient at Edinburgh Royal Infirmary on 13th April 1894.

Complaints.—General weakness ; stiffness and coldness of the hands ; pains in shoulders, wrists, fingers, hips, and knees.

Previous History.—A healthy man until eighteen months ago, when fingers began to feel cold and dead ; he thought they were frost-bitten. The coldness of the fingers was first noticed after curling and lying out in the cold all night while intoxicated. Before the onset of the disease was a robust, healthy man.

Family History.—Good.

State on Admission.—Hands feel heavy, dense, hard, and cold ; skin of hands and fingers hide-bound ; little and ring fingers in each hand contracted ; skin of palms hard, dense, cold, and clammy ; fingers pointed. Hands get blue on the least exposure to cold. Movements of fingers and wrists restricted. Sores on knuckles of little, ring, and forefinger of right hand, and on little finger of left hand ; these sores have been present for twelve months and have not healed. Skin of face looks thin and drawn. Skin of body generally is so much pigmented that the doctor (Dr Reid) who sent the patient to the Infirmary suggested that he might be suffering from Addison's disease.

No visceral disease.

Sensations to heat, cold, and touch normal over the body, though

CASES OF DIFFUSE SCLERODERMIA

No.	Initials.	Age.	Sex.		Occupation.	Hospital Practice.		Private Practice.	Part Affected.					Complications.	Treatment.	Result.
			M.	F.		In-P.	Out-P.		Hands.	Feet.	Face.	Thorax.	Abdomen.			
1	Miss F.	65	..	1	None.	..	..	1	1	1	..	..	..	Rheumatic pains in hands, feet, knees, etc.	Warmth; iodide of potassium.	Died 5 years later from cystitis.
2	D. K.	36	1	..	Stone-mason.	..	1	..	1	1	1	1	..	Pigmentation of skin; muscular pains in fingers, wrists, shoulders, knees, and hips.	Warmth; iodide of potassium; thyroid extract; arsenic; opium.	Slight temporary improvement. Died 2½ years later from pericarditis.
3	D. M.	43	1	..	Stone-mason.	..	1	..	1	..	..	..	..	Sores on knuckles.	Warmth; morphia.	Some improvement.
4	A. H.	44	1	..	Coal-miner.	1	..	..	1	..	..	..	..	Hard swelling (? osteo-arthritis) head of right ulna, and pain and swelling of metacarpal and phalangeal joints.	Warmth; alkaline baths; menthol oil.	*I. S. Q.*
5	R. M.	35	1	..	Stone-mason.	1	..	..	1	1	1	..	..	Scars on knuckles; rheumatic pains in shoulders, elbows, and hips; pigmentation of skin; telangiectases, etc.	Warmth; thyroid extract; quinine; cod-liver oil.	Marked temporary improvement.
6	J. M'I	57	1	..	Merchant.	..	..	1	1	1	1	1	1	Gangrene of tip of forefinger exactly resembling the lesion of Raynaud's disease.	Warmth; arsenic; iodide of potassium.	Died 15 months later—sudden pulmonary œdema.
7	J. D.	26	1	..	Stone-mason.	1	..	..	1	..	1 ?	..	..	Sores on knuckles.	Warmth; fibrolysin.	Marked improvement.
8	J. L.	46	1	..	Coppersmith.	1	..	..	1	..	..	..	..	Rheumatic pains and creaking in shoulders, ankles, and thighs.	Warmth; fibrolysin.	Marked temporary improvement.
9	J. S.	55	1	..	Stone-mason.	1	..	..	1	1	1	1	..	Creaking in wrists, elbows, knees, and ankles.	Warmth; fibrolysin.	Marked improvement. Died 8 months later.

Note.—In all cases massage and passive movements were employed.

somewhat impaired over the hands. Urine has never been porter-coloured.

Treatment.—To keep parts warm; avoid exposure to cold; iodide of potassium.

Progress.—13th June 1894, seen again at Edinburgh Royal Infirmary much *in statu quo*. Iodide to be discontinued; thyroid extract (5 grains, three times daily) substituted.

18th July 1894.—Thyroid treatment produced little or no improvement although the dose was doubled by Dr Reid. The patient says he felt better under the iodide than the thyroid.

19th July 1895.—In April last had an attack of pleurisy with effusion; the chest was tapped. The sclerodermia is more marked; face more affected; cannot close his lips so as to hold his pipe. Skin over the body generally, and especially over the upper part of the chest (manubrium sterni), affected. Feet and legs affected to some extent, much less than the hands and arms. Ulcers on knuckles still from time to time present; the least injury, he says, leads to the formation of a sore.

Has tried arsenic and opium in addition to the thyroid and iodide. The only thing which does him good, he says, is keeping the parts warm. Antimony and the constant current suggested.

The patient died on *26th October* 1896. The immediate cause of death was pericarditis. Dr Reid informs me that the sclerodermia gradually increased and became very marked on arms, face, and chest; the movements of the chest-wall were much impaired and painful. Dr Reid suggests that the sclerodermatous condition of the chest-wall was perhaps concerned in the production of the pericarditis.

Case III.—D. M., aged 43, stone-mason, seen as an out patient at the Edinburgh Royal Infirmary on 28th January 1895.

Complaint.—Coldness and stiffness of the hands.

Duration.—Three years.

Apparent Cause.—Exposure to cold. Fingers became so cold at his work that he was unable to hold the chisel, and the hammer fell out of his hand. Soon after the condition of coldness and stiffness in the hands developed, sores appeared without any apparent cause on some of the fingers; there was little or no pain; the nail of the right forefinger came off.

State on Admission.—Both hands cold, dense, hard, blue or whitish-blue if exposed to cold; skin of palms dense, hard; skin of fingers smooth, tightly stretched, cannot be pinched up. Scars of

old ulcers on some of the knuckles and finger-tips. Movements of fingers much impaired.

Sensibility to touch, heat, and cold normal over body generally, slightly impaired over the hands. Urine never dark in colour.

No visceral disease.

Treatment.—Warmth; liquor morphinæ hydrochlor. (4 minims, subsequently increased to 8 minims, three times a day.)

23rd February 1895.—Slightly better; hands do not feel so cold; sores healed.

Subsequent Progress of the Case.—Not known.

Case IV.—A. H., aged 44, married, coal-miner, admitted to the Edinburgh Royal Infirmary on 29th June 1897.

Complaints.—Stiffness of the fingers and wrists, pain and swelling in the fingers of both hands, more especially the right.

Duration.—Right hand four years, left hand two months.

History.—Before the present condition developed enjoyed good health. About four years ago was dragged on his hands and knees by a waggon for six yards; the right hand was injured; the back of the hand became swollen, the swelling extended to the wrist and metacarpo-phalangeal joints. There was considerable pain in the wrist and back of the hand and fingers, burning in character, with stiffness and soreness of the joints. Was off work for three months. After recovery worked for twelve months; the condition then recurred without obvious cause, and he was again off work for three months; since then has worked two or three days a week. Two months ago the left hand became similarly affected, and he has been unable to work. Has been exposed to cold and weather going to and returning from his work.

State on Admission.—Fairly well nourished; not very muscular; has got much thinner during the past four years.

The morbid appearances are entirely confined to the hands and wrists. The right hand is much more affected than the left. Both hands have a waxy, smooth appearance; the skin feels very hard, is tacked down to the subcutaneous tissue and cannot be pinched up. Hands feel very cold, and the patient states that at times he feels the cold very severely. The movements of the hands and wrists are much impaired.

There is a hard swelling over the head of the right ulna, the metacarpal and phalangeal joints are swollen and somewhat painful, the nails are brittle and speckled over with white spots, the palm

and fingers are contracted. The muscles of the forearm, more especially the extensor muscles, are somewhat atrophied.

The patient states that at times reddish spots appear on the back of his hands, and last for four or five days; the spots are circular in shape and about one-third of an inch in diameter. There is no visceral disease.

Treatment.—Warmth; alkaline baths and menthol oil were applied to the hands with passive movements.

Result.—Some improvement in the movements of the fingers and wrists and disappearance of the pain on movement; little change otherwise.

Discharged 15*th July* 1897.

Case V.—R. M., aged 35, married, stone-mason, admitted to Edinburgh Royal Infirmary on 8th October 1898.

Complaints.—Coldness and stiffness in hands and feet, stiffness in legs, stiffness in face.

Duration.—Two years.

Previous History.—Enjoyed excellent health until two years ago when he began to suffer from cold hands and feet; a year ago noticed stiffness in the legs and arms. Works in the open air and is much exposed to the weather.

Home surroundings good. *Family history* excellent.

State on Admission.—Healthy looking man, but muscles rather wasted. Height, 5 ft. 9 ins.; weight, 10 st. 1 lb. Skin of whole body dark in colour, smooth, and dry. Nipples not pigmented. Hands, feet, forearms, legs, shoulders, and face markedly affected with diffuse sclerodermia.

The condition began in the hands. The face was affected twelve months ago.

The hands feel heavy and cold, like frozen india rubber; they become markedly blue on exposure to cold. The points of the fingers are glazed and shrunken, fingers slightly flexed into the palm; the skin is thin and tightly stretched over the hands and fingers, which are pointed; the nails are long and narrow; there is a transverse groove in the nail of the forefinger of the right hand and a longitudinal groove on that of the little finger of the left hand.

The movements of the fingers and wrists are markedly impaired.

The skin of the forearms is markedly affected; skin of the upper arms normal; skin over both shoulders markedly affected. He cannot raise his arms above his head. Face is markedly affected, skin tightly stretched and feels stiff, especially over the nose, cheeks,

and lower part of the forehead; says he has difficulty in opening the mouth, as in yawning, owing to the tightness of the skin. The ears are much affected, blue and cold; skin thin, glazed, and tightly stretched.

The feet are similarly affected to the hands but in a much less marked degree.

The feet became affected two months later than the hands; they became cold and numb, and he did not feel the ground when they were cold; he then began to complain of weakness and stiffness in the legs, and consequent difficulty in walking. He gradually got weaker and weaker until he was unable to walk. The skin of the legs is slightly affected, the calves feel tight and stretched, and are apt to pain him in walking. The skin of the abdomen, thorax, and thighs is unaffected.

The skin of the body generally is yellowish-brown in colour (markedly pigmented).

He complains of numbness in the fingers and feet when exposed to cold. There are no objective disturbances of sensation.

The tendon reflexes are somewhat diminished; superficial reflexes all distinct.

Scars, the result of old ulcers, which he says were very slow in healing and which developed spontaneously, are present on the metacarpo-phalangeal joints of the 1st, 2nd, and 4th fingers of the right, on the 1st and 2nd fingers of the left hand, on the distal phalangeal joints of the 1st and 2nd fingers of the right hand, and on the 1st, 2nd, 3rd, and 4th fingers of the left. The sores on the fingers began to develop twelve months after the disease commenced.

Three scars are present on the sole of the left foot and two on the left ear. These scars exactly resemble the lesions which are seen in Raynaud's disease and in some cases of syringomyelia.

The patient has never suffered from dead fingers nor passed dark, porter-coloured urine.

Blood—red corpuscles 3,800,000 per cubic millimetre, whites 5800, hæmoglobin normal. Blood film shows nothing abnormal. No visceral disease.

Treatment.—Warmth; thyroid extract, quinine, and cod-liver oil.

Result.—Temporary improvement. Discharged 7th January 1899.

Readmitted 8th February 1899 and discharged 15th February 1899, *in statu quo.*

Seen as an out-patient *12th August* 1903. Is worse. The terminal phalanges of the fingers are permanently flexed; face very pinched; complains of pain and difficulty in swallowing. The veins over the chest and arms are very prominent. No definite signs of aneurysm or intra-thoracic growth.

Seen at Chalmers Hospital on 4th February 1914.—His condition is markedly changed since last seen ten and a half years ago. The sclerodermatous condition is less marked, but the fingers of the right hand are now much more contracted. The slightest exposure to cold makes the hands turn blue; he says that the hands are painful when cold.

The fingers of the right hand are flexed into the palm and cannot be straightened; the palm feels hard, dense, and contracted (Figs. 1 and 2). The tip of the right thumb is ulcerated, the nail is partly destroyed and detached. This ulcer has been present since July; it is now healing. Before the ulcer developed, the tip of the thumb became black. The nails of the 1st, 2nd, and 3rd fingers of the right hand are very long, thin, narrow, pointed, and curved. The nail of the 3rd finger is absent; it was detached recently as the result of a sore which formed on the end of the finger. The skin on the back of the right hand is thin, but is not tied down and fixed; the condition of the back of the hand is very different to what it was ten years ago. There are some spots (telangiectases), small in size, on the back of the right hand.

The left hand is less affected than the right; most of the fingers can be partly straightened (extended). The little finger is rigidly flexed, skin very thin and shiny, the nail very long and narrow. The nails of the other fingers of the left hand are curved. The skin of the fingers is tightly stretched. On the back of the left hand the skin is fairly normal as compared with that of the right.

The face is thin; he can open his mouth much more freely than he used to do. The sclerodermatous condition of the face has to a large extent disappeared.

Numerous bright red vascular spots (telangiectases) are present on the forehead, nose, and cheeks (Fig. 3); they are of various shapes. On pressure the colour disappears from these spots. There are a few small red spots on the tongue, more especially on the right side. The appearance of the face is suggestive of xeroderma pigmentosa.

The skin of the ears is thin and tightly stretched.

Over the neck and upper part of the thorax multitudes of small

FIG. 1.—Right hand in the case of diffuse sclerodermia described in the text, showing ulceration of the tip of the right thumb and contraction of the fingers.

FIG. 2.—Right hand in the case of diffuse sclerodermia described in the text, showing contraction of the fingers.

PLATE I.—TO ILLUSTRATE ARTICLE BY DR BYROM BRAMWELL.

FIG. 3.—Case of diffuse sclerodermia described in the text, showing the vascular lesions on the face, neck, and upper part of the chest.

PLATE II.—TO ILLUSTRATE ARTICLE BY DR BYROM BRAMWELL.

arborescent vessels are seen, red in colour. Over the front of the chest, more particularly over the left side, there are several red vascular areas, not so bright as on the face, distinctly arborescent; on pressure the colour disappears from these spots.

There are a few vascular red spots over the back.

Feet are slightly swollen, the skin is hard, and they get blue on exposure to cold. From time to time he has had sores on the back of the toe joints.

The skin of the abdomen is natural.

The thyroid cannot be felt; it is apparently atrophied. The nipples are not pigmented.

His general health is, he says, good; he sleeps well. His appetite is fair, his bowels regular.

There is no difficulty in making water, but every now and again, perhaps for a fortnight at a time, it comes away without his knowing it and he wets himself.

The difficulty in swallowing which he complained of when last seen ten years ago, lasted for some years and then disappeared; there has been no difficulty in swallowing for the last seven years.

He is thin; the muscularity of the arms is poor, of the legs fairly rood. He says that he sweats very little.

CASE VI.—Mr M'I., aged 57, merchant, seen in consultation on 7th January 1906.

Complaints.—Stiffness of hands, arms, face, chest, lower part of abdomen, and thighs.

Duration.—One year.

Apparent Cause.—None.

Condition when Seen.—Skin over the whole body is hide-bound except over the calves; the hands are more affected than any other part. Skin pale, cold, dense, tightly stretched over the fingers, which are pointed. The patient states that he feels the cold very much; his general health is good. There is some numbness in the hands and feet, and at times a dull aching pain.

No visceral disease. The urine has never been dark coloured. No symptoms of Raynaud's disease. Nothing abnormal detected in connection with the nervous system. Sensation to touch, pain, heat, and cold normal.

Treatment.—Arsenic and iodide of potassium.

Result.—Patient remained much *in statu quo* until 1*st April* 1907, when he was attacked with sudden dyspnœa and the expectoration of blood-tinged, copious, frothy sputa, and died in ten minutes.

The condition was obviously acute pulmonary œdema in a patient affected with sclerodermia. For some days before the attack of dyspnœa he had complained of headache and sickness, but there was no elevation of temperature.

During the year 1900 the tip of the forefinger of the left hand became gangrenous—resembling the lesion seen in Raynaud's disease.

CASE VII.—J. D., aged 26, stone-mason, admitted to Edinburgh Royal Infirmary on 23rd February 1900.

Complaint.—Numbness, stiffness, and coldness of hands.

Duration.—One year.

Apparent Cause.—Exposure to cold during work. Right hand first affected. Patient says he had to cycle to his work, a distance of five miles, in cold frosty weather, and the metal handles of cycle used to be exceedingly cold; this, and holding a cold chisel in his hand, he thinks, is the reason why the hands became affected first.

State on Admission.—Hands of a dead leaden violet colour, on exposure become much more blue. Skin of the hands feels firm, dense, and very cold; it resembles a frozen corpse or a piece of frozen india-rubber. Palms feel cold, hard, and moist. Skin tightly stretched over the fingers and devoid of wrinkles; fingers are pointed. Movements of fingers and wrists are considerably impaired; parts are hide-bound. Patient cannot close his fist. Joints of fingers somewhat enlarged. Three sores, two at the base of the nails of first and second fingers of left hand, one on the extensor aspect of the metacarpo-phalangeal joint of right thumb, are present.

Skin of forearms and face affected.

Says that he is very sensitive to cold; has never passed dark-coloured urine.

No visceral disease. Nervous system normal.

Treatment.—Warmth; thiolysin injections.

Result.—Marked improvement; the patient was discharged on 30th May.

As a result of the thiolysin treatment marked elevations of temperature occurred.

The preparation used was a solution of thiolysin (200 milligrams of thiosinamine (Merck) and 200 milligrams of salicylate of sodium dissolved in 3 c.c. distilled water). Of this solution 30 minims containing 120 milligrams of thiosinamine were injected into the upper arm; the injection was repeated every second day.

The first seven injections were not attended by any alteration in temperature and pulse; after this, each injection was followed by a very considerable rise in temperature and pulse. *Between 14th March and 3rd April* the highest temperature noted was 102.4° F. The injection which was made on 3rd April (which consisted of 20 instead of 30 minims of the solution) was followed by a temperature of 103.4° F., and a pulse of 112.

After this considerable rise in temperature the fibrolysin injections were discontinued—*from 4th to 14th April inclusive* no fibrolysin was given; during this period the temperature and pulse were, practically speaking, normal.

The question arose whether these rises in temperature were the result of septic poisoning (septic decomposition which had occurred in the solution of fibrolysin which was being employed). Consequently a fresh solution (Merck's) in sealed glass capsules was obtained. Of this solution 50 minims (containing 200 milligrams of thiosinamine) were injected *on 15th April*; this was followed by a still greater rise of temperature, viz. 104.4° F.

The fact that the injection of a different and aseptic solution of fibrolysin was also followed by very marked rises in temperature negatived the supposition that the rises in temperature which followed the injections of the solution first employed were due to septic poisoning. This view was corroborated by the fact that in two other cases in which the same solution of fibrolysin as that which produced the rises in temperature in the case of sclerodermia was used no temperature or pulse alterations resulted from the injections.

On 19th April 16 minims of the original solution, containing 60 milligrams of thiosinamine, were injected; this was again followed by a very high temperature (104.2° F.). *On 22nd April* 8 minims of the original solution, containing 30 milligrams of thiosinamine, produced a temperature of 103.8° F.

On 24th April distilled water was injected instead of the fibrolysin. The patient was not, of course, told that the nature of the injection had been changed. There was no rise of temperature. The possibility that the elevations in temperature were merely nervous was consequently excluded.

On 26th April 4 minims of the solution, containing 16 milligrams of thiosinamine, produced a temperature of 101° F. *On 28th April* 20 minims were followed by a tenperature of 102° F.; and *on 3rd May* 20 minims were followed by a temperature of 101° F.

The injections were then discontinued until *18th May*. The

temperature again remained normal during the period that the fibrolysin was not employed.

On 18th May 15 minims of the solution produced a temperature of 102° F.; *on 22nd May* 15 minims produced a temperature of 99.4° F.; *on 25th May* 30 minims of the solution were not followed by any rise of temperature; *on 27th May* 40 minims produced only a temperature of 100° F.

From these statements it will be seen:—

1. That the first seven injections produced no temperature alteration.

2. That a marked rise in temperature followed each dose of the remedy which was subsequently given, different solutions being from time to time employed.

3. That the patient seemed to become accustomed to the remedy; on 27th May a large dose—40 minims—produced a comparatively slight effect.

In Figs. 4 and 5 the alteration in temperature and pulse which followed the injections of fibrolysin are shown.

In Fig. 4 the course of the temperature, taken every hour for twenty-four hours after an injection of fibrolysin, is shown.

At 9.15 P.M. on 28th April 80 milligrams of thiosinamine were injected; this was followed by a slight fall. Then at 3 A.M. (on 29th April) a marked and sudden rise occurred, the temperature reaching 103° F. at 7 A.M.; after this there was a gradual fall, the temperature becoming normal at 11.30 P.M. on 29th April.

Several observations of this kind were made; in all of them the temperature curves were practically the same as that represented in Fig. 5.

At the time this patient suffering from scleroderma was being treated, the same solution of fibrolysin was given subcutaneously to several other patients in the wards. In one of these a very marked elevation of temperature resulted after an injection. The patient, a man aged thirty-four, was suffering from disseminated sclerosis.

On 18th, 22nd, 23rd, 26th, and 28th April 20 minims of the same solution as was used in the stone-mason's case were injected, without any elevation of temperature being produced.

On 2nd May 30 minims of the same solution produced a very high temperature—105.8° F.

Why the injection on 2nd May (although it was rather a larger

dose than had been previously given—30 instead of 20 minims of the solution) should have been followed by such a marked temperature disturbance, while the previous injections produced no temperature alteration, I am unable to say.

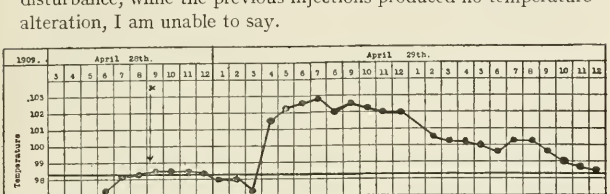

FIG. 4.

One of my former house-physicians wrote me on 12th May 1909, saying: "I happened to mention to the house-surgeon here (Paddington Green Children's Hospital, London) that in one of

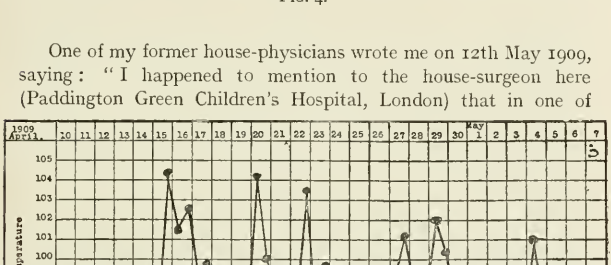

FIG. 5.

your cases of sclerodermia a sudden rise of temperature occurred after the injection of fibrolysin. He tells me he had once a case (not a case of sclerodermia) in which a similar rise of temperature took place."

CASE VIII.—J. L., aged 46, coppersmith, admitted to Edinburgh Royal Infirmary on 3rd March 1911.

Complaints.—Stiffness of hands, pains in hands, shoulders, thighs, and knuckles.

Duration.—Three months.

History.—Stiffness in hands first felt three months ago—he found he could not grip his hammer and chisel properly; at this time there was some stabbing pain in the palms and fingers. Had to give up work six weeks ago. Has also complained of pains in shoulders, thighs, and ankles. Until this illness commenced was a healthy man.

Family History.—Unimportant.

Social Condition.—A coppersmith. Has to put his hands in cyanide of potassium, muriatic acid and vitriol. Works a good deal with a hammer and cold chisel.

Condition on Admission.—Small man, well built, healthy looking. Hands and fingers slightly swollen; skin hard, shiny, tightly stretched, cannot be pinched up; some tenderness on pressure over the palms; movements of fingers much impaired. The sclerodermatous condition is confined to the hands. The patient complains of aching pains in shoulders, ankles, and thighs; on movement, creaking is well marked in shoulders and ankles.

No visceral disease. No evidence of any disease of the nervous system. Urine has never been dark coloured.

Treatment.—Warmth; fibrolysin injections.

Result.—Decided improvement in condition of hands.

16th April.—Discharged distinctly better.

6th June 1911. Readmitted. Pains in ankles; knees and shoulders worse; some swelling of wrists. Sclerodermatous condition of the hands is much *in statu quo*.

Treatment.—Hot-air baths; guaiacol carbonate, 5 grains, three times daily.

19th June.—Discharged improved.

Case IX.—J. S., aged 55, stone-mason, admitted to Chalmers Hospital on 10th June 1913.

Complaints.—Coldness and stiffness of hands; stiffness of face, arms, chest, legs; shortness of breath on exertion; occasional attacks of sickness and diarrhœa.

Duration.—One year.

Apparent Cause.—Exposure to cold.

History.—The numbness in the hands was first felt after clearing out some cold spring water from a drain.

July 1912.—Attack of pleurisy; in bed four or five days (con-

fined to the house a fortnight). After this hands got worse ; after exposure to cold became blue and discoloured. Stiffness of fingers developed ; feet and toes became similarly affected (cold and stiff). About *Christmas* 1912 lost his appetite and was troubled with stomach ; vomited occasionally. Rheumatic pains in ankles, legs, shoulders, and chest developed in *March* 1913 ; there was also occasional looseness of the bowels. Until the present illness commenced was a very healthy man.

Family History.—Unimportant.

Condition on Admission.—Slightly-built man. Height, 5 ft. 4 ins. ; weight, 9 st. 3 lbs. Expression of face immobile. Marked dryness of skin of body and of hair. Does not perspire except over the hands and feet, which feel cold and moist.

Marked sclerodermatous condition present on hands, arms, face, chest, scalp, buccal mucous membrane, and feet.

The hands are most markedly affected ; after the slightest exposure to cold they become blue. Hands feel heavy, hard, and dense ; skin lardaceous, tightly stretched, thin over the fingers, which are pointed ; skin cannot be pinched up over the hands, front of the chest, scalp, thighs, and feet. Movements of parts greatly impaired. The cheeks feel brawny.

On movement, creaking felt in elbows, wrists, fingers knees, ankles, and toes.

No visceral disease. Blood normal. No evidence of disease of the nervous system. Urine normal ; never has been porter-coloured.

Sensation to touch, pain, heat, and cold normal all over the body.

Treatment.—Rest in bed, warmth, light diet ; fibrolysin injections, thyroid extract, iodide of potassium, and arsenic.

On 16th June thyroid extract (5 grains twice daily, increased to 5 grains three times daily, on 22nd June) was prescribed. *On 3rd July* the thyroid extract was discontinued.

On 4th July iodide of potassium (10 grains three times daily) and arsenic (2 drops of the liquor arsenicalis) prescribed.

Result.—Very marked improvement. Sclerodermatous condition of skin became markedly less, movements of affected parts much freer.

9th September 1913 discharged very much improved.

Elevations of Temperature following Injections of Fibrolysin.—In this case the same alterations in temperature and pulse which were seen in case vii. followed some of the injections of fibrolysin.

In several instances a blood count was made immediately before

an injection, and during the subsequent rise of temperature. The only alteration found was a slight diminution of the red corpuscles, a decided increase in the polymorphs, and a diminution in the small mononuclear white corpuscles.

Additional Note.—Patient died suddenly of heart failure on 23rd February 1914; post-mortem examination showed a condition of chronic interstitial myocarditis.

Discussion

The Vice-President said the Society always expected interesting material from Dr Byrom Bramwell, and he did not think they were ever disappointed, because he either brought forward cases from his wide clinical experience, or brought rare cases with new methods of treatment. He now brought forward a disease which must be more or less unfamiliar to the great majority present, and he had described the very marked benefit which had resulted from fibrolysin treatment. It was not a paper which would give rise to much discussion, but they would be glad to hear the remarks of any members of the Society.

Dr Cranston Low agreed that the Society was very much indebted to Dr Bramwell for having brought these cases forward. There was no doubt the condition was a very rare one, and personally he had only seen three cases. With regard to the question of occupation his experience did not have any bearing on that point, as all his patients had been females. There had been within the last ten years only 2 cases of diffuse sclerodermia in the Skin Wards of the Royal Infirmary, and both cases were females. That did not include another case of acute sclerodermia, which was generally known as sclerœdema, which began with an erythematous condition, became sclerosed, and ran a course of about three months.

As to the question of fibrolysin, Dr Bramwell said that after the first few injections there was no rise of temperature; was it not possible to explain that fact by the sensitisation of the patient to the fibrolysin—anaphylaxis? In cases where repeated injections are administered, a number have to be given before the patient becomes sensitised to the drug. Were there any signs of erythema, such as were seen after diphtheritic antitoxin? If that supposition were correct one would expect that the first dose would not give any rise of temperature, and that the later ones would.

The first patient shown was very extraordinary, and showed well how these cases led to an extremely atrophied condition of the skin.

Dr Douglas Guthrie was much interested in the paper, having had the opportunity of seeing a case of diffuse sclerodermia shown by Dr Hunter a year ago, at the Glasgow Medico-Chirurgical Society. The patient was a woman, and the appearances were very much the same as the first patient shown by Dr Bramwell. The condition was, however, associated with a peculiar condition of the fingers, consisting of subcutaneous nodules, the skin over which ulcerated from time to time, and a calcareous pellet dropped out. He believed that at least one other case of this kind had been published in the *British Journal of Dermatology*. Dr Bramwell's experience as regards sex was unusual, the universal experience being that most of the cases occur in females.

Dr R. A. Fleming had only one or two remarks to make, having seen few cases of the disease. In the first place, he presumed Dr Bramwell associated massage with the use of fibrolysin. Personally he did not think much benefit would result from fibrolysin in any form unless combined with massage. One if not two of the cases he had seen were cases of Dr Bramwell's, but one was a case of Prof. Greenfield's, which was temporarily under his care. In that patient there was also a very extensive amount of thickening of the muscles and tacking down of the skin to the muscles pretty much all over the body. That patient certainly improved a little with fibrolysin, associated with general massage.

Dr Fleming asked if Dr Bramwell in the cases which died got any statistics about the post-mortem findings? He (Dr Fleming) some years ago had the opportunity of examining a case of Raynaud's disease which accidentally died. That patient presented no thickening of the skin at all. In the nerves of the affected parts—the hands and feet—there was very marked thickening of the vessels, especially in the middle coat of the smaller arteries, and this somewhat curious thickening could be traced also in similar vessels in the cord. There was no obvious change in the nerves, in the tracts, or in the cells of the cord. He wondered if there might not be thickening in the vessels of the skin in cases of sclerodermia.

Dr Byrom Bramwell, in reply, said he was glad his paper had

interested the Society, and thanked the President and other speakers for their remarks.

In all his cases warmth seemed to do good—in all the cases massage and passive movements were employed when the joints began to get more supple, but he must say that in the cases in which fibrolysin was employed the improvement was infinitely greater than in the cases in which it was not; he was consequently disposed to attribute a large part of the improvement to the fibrolysin itself. He could not answer Dr Fleming's question; he had not had the opportunity of making a post-mortem in any of the fatal cases.

With regard to Dr Low's question, there was no erythema such as one gets after diphtheritic antitoxin. A series of blood counts was made, and it was found that the only change was a very slight diminution in the reds, a decided increase of the polymorphs, and a diminution in the small mononuclear leucocytes.

Dr Bramwell would like to hear what Dr Low thought about the diagnosis of xeroderma pigmentosa.

Dr Low remarked that it was not uncommon to get considerable dilatation of the vessels in any atrophic scar, and especially on the hands and face where the skin was exposed to the light, but not so marked as in the patient which Dr Bramwell had shown.

Meeting VI.—March 4, 1914

Dr John Playfair, *President*, *in the Chair*.

I. Election of Members.

George Reginald Edward Gray Mackay, M.B., Ch.B., 5 Walker Street, and Edmund Ivens Spriggs, M.D. Lond., F.R.C.P. Lond., Duff House, Banff, were elected Ordinary Members of the Society.

II. Exhibition of Model.

Dr J. K. Miller Dickie and *Dr J. S. Fraser* exhibited reconstruction model of middle and inner ear.

III. Original Communications

1. LABYRINTH SUPPURATION

By J. S. Fraser, M.B., F.R.C.S., Edinburgh; Senior Assistant-Surgeon, Ear and Throat Department, Royal Infirmary, Edinburgh; Aural Surgeon, Leith Hospital; Lecturer on Diseases of the Nose, Throat, and Ear, School of Medicine of the Royal Colleges, Edinburgh

Development and Anatomy of the Labyrinth

The membranous labyrinth is developed from the otic vesicle—a hollow ingrowth of epiblastic neuro-epithelium, which sinks into the mesoblast. Later the otic vesicle becomes differentiated into (1) the Pars Superior, or Static Labyrinth, consisting of the saccule, utricle, and the three membranous semicircular canals: to this part is attached the vestibular nerve and its ganglion. (2) The Pars Inferior, or Membranous Cochlea, to which the cochlear nerve and its spiral ganglion are attached. From the point of view of comparative anatomy the static labyrinth is considerably older than the auditory labyrinth.

1. *Pars Superior.*—The three membranous canals communicate with the utricle by five openings—the superior and posterior vertical canals having a common opening (*crus commun*) at their non-ampullated ends. The three canals lie in the three dimensions of space: each canal is dilated at one end into an ampulla which contains a small mound of neuro-epithelium called the crista. This mound is capped by the cupola, and is supplied by a branch from the vestibular nerve. The utricle itself shows a layer of epithelium on the inner side of its external wall; the neuro-epithelium on the saccule is placed on the antero-internal wall of the cavity at right angles to the plane of the sensory epithelium in the utricle. The utricle and saccule each give off fine ducts which join to form the endolymphatic duct; this again ends in the endolymphatic sac between the layers of the dura on the posterior surface of the petrous pyramid. The utricle and saccule are supplied by the vestibular nerve and its ganglion in the internal auditory meatus.

2. *The Pars Inferior, or Membranous Cochlea,* is a blind tube (somewhat triangular on section), coiled round a central stem or modiolus. In the floor of this tube, which is wider at the apex than at the base of the cochlea, lies Corti's organ—a small mound of neuro-epithelium supplied by the cochlear division of the eighth

nerve and its spiral ganglion, which is situated in the modiolus. In the embryo, and probably also in the adult, the narrowest part of the cochlea is connected to the saccule by means of a fine duct (*canalis reuniens*).

The Bony Labyrinth.—In the early embryo the membranous labyrinth is surrounded by a layer of mesoblast, which develops into a hollow capsule of cartilage. This, later on, becomes converted into dense bone and embedded in the petrous temporal; the cartilage bone capsule of the labyrinth can, however, always be distinguished from the surrounding lamellar bone, with its marrow spaces. If this cartilage bone capsule be artificially cut out by a skilled workman, it will be found to consist of (*a*) the bony semicircular canals, which contain the membranous canals, and open into (*b*) the bony vestibule which contains the utricle and saccule, and communicates in front with (*c*) the bony cochlea, which encloses the membranous cochlea. On the outer wall of the vestibule we have the oval window which is closed in the living by the footplate of the stapes and the annular ligament. The bony cochlea resembles the shell of a snail, and lies with its base towards the internal auditory meatus. From the central stem or modiolus of the cochlea the bony spiral lamina projects into each coil of the bony cochlea. To the edge of the bony spiral lamina the membranous cochlea is attached. The outer surface of the membranous cochlea is attached to the outer wall of the bony cochlea. In this way each coil of the bony cochlea is divided into three parts: (1) the scala vestibuli above, which communicates with the vestibule, (2) the scala tympani below, which begins at the round window, and (3) the membranous cochlea with its spiral lamina, which lie between (1) and (2). At the apex of the cochlea the scala vestibuli and the scala tympani become continuous with one another at the helicotrema. The membranous labyrinth is filled with fluid (endolymph), and is surrounded more or less by fluid (perilymph), which separates it from the endosteal lining of the bony labyrinth.

The best way to understand the anatomy of the labyrinth is to think of a tunnel driven through a small hill. If a section be made through this hill, the cut end of the tunnel will be seen, and round it the stonework forming the wall of the tunnel. The hill corresponds to the petrous bone, the stone walls of the tunnel to the dense cartilage bone which forms the true capsule of the labyrinth. The cavity of the tunnel corresponds to the hollow space within the bony labyrinth. If we further suppose that the tunnel

contains a train, and that both tunnel and train are flooded with water, we find that the water in the train corresponds to the endolymph, the train itself to the membranous labyrinth, and the fluid between the train and the walls of the tunnel to the perilymph.

The outer wall of the bony labyrinth is a party wall, *i.e.* it is also the inner wall of the middle ear. For this reason the oval and round windows, the external or horizontal canal, and the promontory, which contains part of the cochlea, are exposed to infection in cases of otitis media. The oval window communicates with the perilymph space of the vestibule, while the round window, which lies at the top of a deep niche, communicates with the perilymph space of the cochlea. Vibrations of the perilymph, set up by movement of the footplate of the stapes in the oval window, pass up the scala vestibuli of the cochlea to the apex, and then through the helicotrema down the scala tympani to the round window. Occasionally the bony labyrinth is almost entirely surrounded by air cells which communicate with the tympanic cavity and mastoid antrum, and form part of the middle ear cleft.

On the inner wall of the mastoid antrum we find a series of small veins—the remains of an important fœtal vein—which pass through the fossa subarcuata beneath the dome of the superior canal, to reach the posterior surface of the petrous pyramid, and there to open into the superior petrosal sinus. Infection may pass along this route and give rise to extradural abscess and meningitis. This mode of infection has nothing to do with labyrinthitis.

The lymphatic system of the labyrinth is divided into two parts—endolymph and perilymph. The endolymph system ends in the saccus endolymphaticus already mentioned. The perilymph communicates directly with the subarachnoid space of the posterior cranial fossa by means of the cochlear aqueduct, which opens out of the scala tympani of the cochlea in the region of the round window.

The membranous labyrinth and its capsule of cartilage bone are supplied by the internal auditory artery—a branch of the basilar—which enters at the internal auditory meatus. The veins, on the other hand, leave the labyrinth along the aqueducts of the vestibule and of the cochlea.

Physiology

From the physiological point of view the internal ear is divided into (1) *the auditory labyrinth*, and (2) *the static labyrinth*.

1. The blind tube of the membranous cochlea commences within the vestibule, and coils round the central stem or modiolus as described above. At its beginning within the vestibule the cochlear duct is narrow, and at this part the highest tones are appreciated. At the apex of the cochlea the duct is wider, and here the lowest tones are heard. The range of hearing of the human cochlea extends from sixteen double vibrations per second up to about eighteen thousand—at least in young subjects. The auditory apparatus is tested by means of the voice (spoken and whispered), the watch, or akoumeter, a series of tuning-forks ranging from C. 16 up to C. 4096 double vibrations per second. Above this the upper tone limit is best tested by means of the monochord—an instrument on the principle of the violin string, which can be made to vibrate transversely (lower tones, 4000 6000 D.V.S.) or longitudinally for the higher tones, 7000 18,000 D.V.S. It is unnecessary to describe these tests further. In order to establish the presence of complete unilateral deafness (as in cases of labyrinth suppuration) Bárány has invented the clockwork noise apparatus: this instrument is inserted into the patient's good ear, and, when in action, fully occupies the attention of this ear, so that any sounds which the patient hears must be appreciated by the diseased ear. Stopping the healthy ear or rubbing the auricle with the hand serve the same purpose as the noise apparatus.

2. *The Static Labyrinth* (Bárány). Movements of the endolymph in the membranous canals act on the crista and cupula, and the stimuli so created pass by way of the vestibular nerve to the vestibular nuclei at the base of the brain: from these nuclei fibres radiate (*a*) to the nuclei of the motor nerves of the eye muscles (giving rise to nystagmus), (*b*) to the motor neurones of the spinal cord (giving rise to the irregular gait characteristic of vestibular lesions), and (*c*) to the cerebellum. Ewald showed, by experiments on pigeons (application of pressure to an artificial fistula in the horizontal canal), that movement of the endolymph in the right horizontal canal, from the smooth end towards the ampulla, caused horizontal nystagmus to the right, while movement in the opposite direction produced a weaker horizontal nystagmus to the left. In the external canal, movement of the endolymph from the smooth end to the ampulla is therefore the more effective movement. Vestibular nystagmus is of the following type: Horizontal nystagmus to the right consists of a quick twitch of the eyes to

the right and a slower movement to the left—it is a rhythmic associated movement. Vestibular nystagmus is always most distinct when the patient is made to turn his eyes in the direction of the rapid twitch; on the other hand, the nystagmus will be lessened or abolished when the patient looks in the direction of the slow component. It is important to remember that the slow movement of the nystagmus is the real vestibular component; the quick twitch is voluntary, and is abolished when the patient is under chloroform.

The functions of the utricle and saccule are not definitely known. It is believed, however, that they enable us to appreciate passive movement in a straight line when the eyes are closed or when we are in the dark, just as the canals enable us to appreciate passive rotation. It has also been suggested that the saccule may enable us to hear noises as distinguished from musical tones.

Clinical Methods of testing the Vestibular Apparatus (Bárány)

It is first necessary to test the patient for spontaneous nystagmus, as some normal people have slight but equal nystagmus on looking to the extreme right or left.

Rotation.—The patient is placed in a turning-chair with his head upright, and is rotated at the rate of ten complete revolutions in twenty seconds. At the end of the tenth turn the rotation is suddenly stopped, and the duration of the resulting nystagmus is measured by a watch: if opaque spectacles are worn by the patient the nystagmus is rendered more marked. When the head is in the erect position the rotation affects only the two horizontal canals, as they alone are in the plane of rotation. At the beginning of rotation to the right the endolymph in the right canal, in virtue of its fluid inertia, flows towards the ampulla, relatively speaking: in the left canal the fluid flows away from the ampulla. As the rotation is continued, the endolymph catches up with the movement of the canal wall until it rotates at the same rate. If now the rotation of the individual be abruptly stopped, the movement of the endolymph will continue, but its flow relative to the ampulla will be in the reverse direction to what it was at the beginning of rotation. In order to understand the movements of the endolymph and cristæ, we have only to think of what occurs in an Edinburgh tram car—especially on a wet day. When the car starts suddenly,

all the passengers are violently jerked backwards, but, as the car goes on, they again assume an upright position, and maintain it until the car stops suddenly, when they are all jerked forwards. The cristæ of the membranous canals correspond to the passengers.

Nystagmus produced by suddenly stopping the rotation of a patient is known as "after-nystagmus." The average duration of the horizontal "after-nystagmus" in a normal individual, due to rotation with the head erect, is from fifteen to thirty seconds. By bending the head in various directions it is possible to investigate the remaining canals; this, however, is of little practical importance. We must remember that the posterior canal on the right side is on a plane parallel to that of the superior canal on the left side; the same thing, of course, holds with regard to the posterior canal on the left side, and the superior canal on the right side (Crum-Brown).

The laws of nystagmus are as follows: I. Each pair of canals induces nystagmus in its own plane. II. Nystagmus, due to rotation, corresponds to the line of intersection of the horizontal plane with the cornea.

Caloric Nystagmus.—If a patient with normal vestibular apparatus has his right ear syringed with cold water when his head is erect, horizontal and rotatory nystagmus to the left is produced. If warm water (110° F.) be used, the nystagmus is rotary to the right (the reader must distinguish between rotary nystagmus and nystagmus due to rotation). The explanation of caloric nystagmus is as follows: When the head is upright, the summit of the superior canal is the highest point in the labyrinth, while its ampulla lies lower down, near the outer labyrinthine wall. If one cools down the outer wall of the labyrinth by syringing cold water against it, a downward current is set up which acts on the cristæ of the superior and horizontal canals, and gives rise to nystagmus. In the same way warm water causes an ascending current; if the fluid used be at body temperature there is, of course, no reaction. If the vestibular apparatus be destroyed, or the vestibular nerve paralysed, nystagmus cannot be induced by syringing that ear. The caloric method has this advantage over rotation that we can, by syringing, test each ear separately.

When nystagmus to the left is produced in a normal person, he tends to fall to the right if he stands with his feet together and eyes shut, *i.e.* he falls in the direction of the slow component of nystagmus. If the head be turned ninety degrees to the left he

falls forwards; if turned ninety degrees to the right he falls backwards.

Galvanism may be used to test the vestibular apparatus. When the cathode is applied to the right ear we get rotary nystagmus to the right; when the anode is applied we get nystagmus to the left. Galvanism, however, causes nystagmus even if the labyrinth be destroyed, as it acts on the vestibular nerve. For this reason it is not of great practical importance, except in cases of cerebello-pontine angle tumour.

Fistula Symptom.—An olivary ear-piece—attached by rubber tubing to a Politzer bag—is fitted tightly into the external auditory meatus. If the bag be now squeezed so as to raise the air-pressure in the external meatus, nystagmus is produced to the affected side (typical fistula symptom). Occasionally we get a reversed fistula symptom, *i.e.* compression produces nystagmus to the opposite side. The fistula symptom is only obtained in cases in which a fistula exists in the bony wall between the middle and inner ear, and in which the vestibular apparatus is more or less intact.

Pointing Test.—The patient shuts his eyes, closes his hand (palm downwards), and extends his index finger to meet the index finger of the examiner. He is then told to slowly flex the hand at the wrist-joint, and then to raise it again to meet the examiner's finger. Any pointing error present is in the direction of the slow component of the nystagmus. A spontaneous error in the pointing test is usually due to a lesion in the posterior fossa, such as a cerebellar or extradural abscess.

Pathology of Labyrinthitis (Braun and Friesener: Ruttin)

Infection of the inner ear occurs once in every hundred cases of purulent otitis media—usually as the result of an acute exacerbation in a chronic case.

Labyrinthitis may be—(1) *circumscribed*, or (2) *diffuse*.

1. *The circumscribed form* may affect only the bony capsule (para-labyrinthitis), or it may have invaded the perilymph space (peri-labyrinthitis). These two forms may be combined.

Within the vestibule there is a fibrous septum (Alexander), which divides the utricle and canals on the one hand from the saccule and cochlea on the other. In this way labyrinthitis may remain for a period confined to one set of structures. The perilymph spaces of the ampullæ of the canals are almost completely shut off

from the vestibule by septa of connective tissue. Peri-labyrinthitis may, in this way, be further limited.

In the cochlea, labyrinthitis may be limited to the lower half of the basal coil, whether the infection passes by way of the round window or oval window. The lower half of the basal coil of the cochlea remains the lowest portion of the cochlear cavity in every position of the head (Ruttin).

2. *Diffuse labyrinthitis* occurs in two forms: (*a*) Empyema of the labyrinth (combined inflammation of the peri- and endolymphatic spaces), and (*b*) pan-labyrinthitis, in which the bony capsule is affected in addition.

Circumscribed and diffuse labyrinthitis may be acute, subacute, or chronic.

Labyrinthitis may also be classified according to the causal disease or organism, *e.g.* scarlatinal, cholesteatomatous, tubercular, pneumococcic, etc. Fracture of the base of the skull and injury with a pointed instrument, such as a knitting-needle, may give rise to a traumatic form of labyrinthitis.

Lastly, labyrinthitis may be classified according to the route of infection: (1) *Meningeal*, as in cerebrospinal meningitis; from 15 to 50 per cent. of cases of cerebro-spinal meningitis are followed by deafness; (2) *Tympanic*, as in suppurative otitis media; and (3) *Metastatic*, as in mumps.

1. *Meningeal cases.* From the meninges the labyrinth may be infected through (*a*) the aqueduct of the cochlea from the subarachnoid space, (*b*) along the internal auditory meatus, (*c*) by necrosis of the inner bony wall in cases of extradural abscess.

2. *Tympanic.* From the middle ear the infection may pass to the labyrinth by way of (*a*) the oval window; (*b*) the round window; (*c*) fistula of the external canal; and (*d*) fistula through the promontory. If, for example, we take a case in which the promontory of the external canal is gradually absorbed by pressure of a mass of cholesteatoma in a case of chronic middle-ear suppuration, we find that in time the bone is worn through so that the cholesteatoma is now in direct contact with the endosteum lining the canal. A little fibrinous exudation may occur in the canal in the region of this fistula (*circumscribed labyrinthitis*). The condition present in such a case corresponds to that in Ewald's experiment on the pigeon. If the air pressure in the external meatus and middle ear be now artificially raised, pressure will be exerted through the fistula on the labyrinthine fluid, and therefore on the crista of the

external canal; giddiness and nystagmus are thus produced (fistula symptom). The hearing power is not affected, except by the otitis media already present. If now an acute exacerbation of the otitis occur, the feeble barrier remaining may be broken down so that the labyrinth becomes generally infected. At first the inflammatory exudation is sero-fibrinous, with only a few round cells (*diffuse serous labyrinthitis*), and if the process stops at this stage the labyrinth may almost entirely recover. Such a mild attack may occasionally follow the radical mastoid operation. On the other hand, if the inflammatory process continues, the labyrinthine fluid becomes definitely purulent (*manifest diffuse purulent labyrinthitis*): in this condition the patient suffers severely from giddiness, vomiting, and loss of balancing. He is absolutely deaf, but does not usually complain much of tinnitus. Within a few days (3 to 14) the membranous labyrinth is entirely destroyed, and the condition passes into the stage of *latent diffuse purulent labyrinthitis*. This stage may last for several months, while granulation tissue and new bone are formed in the labyrinth. Finally, the hollow spaces of the inner ear become filled with connective tissue and new bone, and the case passes into the stage of *spontaneous cure of labyrinth suppuration with compensation*.

The inflammatory process may extend from the labyrinth—(1) By way of the aqueduct of the cochlea to the subarachnoid space of the posterior fossa, and give rise to lepto-meningitis. (2) The infection may spread to the internal auditory meatus along the subarachnoid sheath of the cochlear nerve, or along the nerve to the saccule or utricle, and give rise to lepto-meningitis. (3) In rare cases pus accumulates in the endolymphatic sac (empyema of the saccus). (4) The pus in the labyrinth may burst inwards, usually through the posterior or superior canals, and give rise to extradural abscess.

Politzer has pointed out that an abscess may form in the fundus of the internal meatus, and may destroy not only the nerve fibres but also the labyrinthine artery: this results in necrosis of the membranous labyrinth, and of a large part of its cartilage bone capsule.

The endosteum which lines the bony labyrinth is the most resistant of all the labyrinthine structures. In those cases of labyrinthitis in which spontaneous cure occurs, the organisation of the pus in the labyrinth proceeds from this membrane: osteoblasts pass in to the granulation tissue which fills the labyrinth and form new bone until, eventually, the hollow spaces of the labyrinth are entirely obliterated.

It will be seen that labyrinthitis may end in (1) return to normal, in cases of mild serous labyrinthitis; (2) spontaneous cure with slight permanent changes; (3) spontaneous cure with destruction of the labyrinthine function; (4) extension to the intracranial structures. If labyrinthitis extends to the cranial cavity it usually gives rise to lepto-meningitis; more rarely it leads to extradural abscesses, cerebellar abscess, or sinus thrombosis. The most frequent path of infection, in cases of meningitis, is the perilymphatic or cochlear aqueduct; if it were not that this canal is very narrow, and that in some cases swelling of its lining membrane completely shuts off the perilymph in the cochlea from the subarachnoid space, otitic meningitis would be even more frequent than it is.

The Clinical Aspect and Treatment of Labyrinthitis (Ruttin)

1. *Circumscribed Labyrinthitis, and Labyrinth Fistula.*

In this condition the patient complains of attacks of dizziness, with occasional vomiting, especially on stooping or turning quickly. Spontaneous nystagmus may or may not be present; if present, it may be to the healthy side, to the diseased side, or even to both. The caloric reaction is usually diminished. With the noise apparatus in the healthy ear, the patient can, as a rule, hear fairly well. The fistula symptom is present (normal or reversed).

Treatment.—The radical operation should be performed, but great care must be taken not to interfere with the fistula. If, as sometimes happens, the attacks of dizziness, from which the patient suffers, interfere with his work, the labyrinth operation may be performed (see page 181).

2. *Diffuse Serous Labyrinthitis*

This condition occupies an intermediate position between circumscribed labyrinthitis and manifest diffuse purulent labyrinthitis.

Symptoms of Diffuse Serous Labyrinthitis.—The patient complains of deep-seated pain in the ear, tinnitus, and deafness. The vestibular symptoms (dizziness, vomiting, and loss of balancing) are more important. The patient lies on the sound side and looks towards the diseased side as, in this position, the nystagmus and giddiness are less. The patient complains that external objects appear to move from the sound to the healthy side. The tem-

perature is not raised. With the noise apparatus in the sound ear the patient is not absolutely deaf. If the lesion be on the left side, spontaneous nystagmus to the right is present; when standing with eyes shut the patient tends to fall to the side of the lesion, *i.e.* in the direction of the vestibular component of the nystagmus—in this case to the left. The vestibular reaction to rotation and syringing are, as a rule, still present, though diminished. The fistula symptom may be present, but is usually absent.

Treatment.—If the patient can still hear with the affected ear, the labyrinth operation should *not* be performed. If, on the other hand, the diseased ear be quite deaf and the vestibular reaction absent, the labyrinth must be opened and drained at the time of the radical mastoid operation; in other words, if we cannot diagnose between diffuse serous and diffuse purulent labyrinthitis, we must treat the case as one of the more dangerous variety.

3. *Manifest Diffuse Purulent Labyrinthitis*

This condition lasts only from three to fourteen days. During this period the patient suffers from intense giddiness, vomiting, and loss of balancing. The vomiting is accompanied by nausea, and is not of the effortless cerebral type. Complete deafness is present when the noise apparatus is going in the sound ear. Marked spontaneous nystagmus is present to the sound side, and the caloric reaction is absent on the diseased side. Spontaneous pointing and falling are to the affected side. The fistula symptom is not present, because the membranous labyrinth is destroyed. The patient is too ill for the rotation test.

Treatment.—If there are no urgent symptoms, such as severe pain, fever, or signs of intracranial trouble, it is permissible to wait in order that a barrier may form. If, on the other hand, interference is called for, a free radical mastoid operation should be at once performed, so as to expose the promontory and the two windows. The dura of the posterior fossa over the sinus should then be laid bare with a blunt gouge. The dura is then separated from the bone of the posterior fossa in an inward direction by means of a blunt dissector, and protected from injury with a small flat spoon or Stacke's protector. The petrous bone is now removed with the gouge and hammer in an inward and forward direction, until the posterior and external canals have been freely opened up behind

the facial nerve. The vestibule is now opened from behind by tracing up the smooth end of the external canal. Finally, the vestibule should be opened in front of the facial nerve by two or three blows with a fine gouge, removing the outer wall of the promontory, and joining the oval and round windows. At the present time the general opinion is that the cochlea should not be further interfered with unless meningitis be present. In the latter case the inner wall of the vestibule should be opened up and the subarachnoid space of the internal meatus drained (trans-labyrinthine drainage of West and Scott).

4. *Latent, Diffuse, Purulent Labyrinthitis*

At the end of from three to fourteen days the manifest stage passes into that of latent, diffuse, labyrinth suppuration. The labyrinth is now entirely destroyed. In this latter stage the patient will, if carefully questioned, give a history of recent dizziness, vomiting, and loss of balancing. These symptoms have, however, now passed off.

Symptoms in the Latent Stage.—There is complete deafness in the affected ear. Spontaneous nystagmus is usually absent, but there may be a slight rotary twitch to both sides. The caloric reaction is absent in the diseased ear. On rotation, there is a marked difference in the duration of the "after-nystagmus," depending on the direction of the rotation, *e.g.* if the patient have a latent labyrinthitis on the right side, and if we rotate him to the right (thus mainly testing the left or healthy labyrinth), he will get horizontal nystagmus to the left of almost normal duration—say twenty seconds. When, however, we rotate him to the left (thus mainly testing the right or diseased labyrinth), we only produce very slight nystagmus to the right of from four to ten seconds duration.

Treatment.—If the radical operation is called for, the labyrinth operation, described above, must also be performed. If this be not done the patient runs a grave risk of getting meningitis as the result of the mastoid operation alone.

5. *Healed Labyrinthitis with Compensation*

This condition is probably present at a period of six months and upwards after the attack of purulent labyrinthitis. The patient is quite deaf in the affected ear, and the caloric reaction is

absent. Rotation, however, to the right and also to the left, produce an equal duration of "after-nystagmus," because compensation has been established, and the external canal on the healthy side reacts equally to both directions of rotation. In such a case, even if the radical mastoid operation be called for, Alexander holds that it is not necessary to do the labyrinth operation, as there is no fear of infection spreading from the inner ear to the meninges. Other otologists, however, consider that it is impossible to be quite sure that the labyrinth is filled with new bone. They believe that pus may still be present, and that, for this reason, it is safer to do the labyrinth operation at the time when the radical mastoid operation is performed.

Discussion

The President said all who had seen Dr Fraser's demonstration must have been impressed with its beauty. Many of them were no doubt rather rusty in their knowledge of the inner ear, and were all the better of the demonstration. The production of such beautiful microscopic specimens must have entailed very great labour and care. This was not a paper which lent itself very much to discussion, there were not many present who were able to speak on it, but one or two were, and the Society would be very glad to hear any remarks.

Dr Logan Turner said he had had more than one opportunity of expressing his admiration of the work which Dr Fraser had done, and did so again along with his appreciation of the value of such work. Personally he could look back to the time when cases were lost after the mastoid operation, death being due to meningitis associated with latent purulent labyrinthitis, and although not very common they did occur. At that time they had no method of examining the function of the inner ear beyond the tuning-fork test, and they were not able to investigate the semicircular canals. Thanks to the work of the young Vienna School and such work as Dr Fraser had done, they were now able to investigate the function of the inner ear before attempting any mastoid operation, and in that way were able to decide whether the disease was confined to the middle ear or affected the inner ear also.

A futher point was the influence of inner-ear disease on intracranial complications. The slides shown demonstrated that many

of these patients died from intracranial complications. So long as the condition was confined to the middle ear, perhaps ½ per cent. to 1 per cent. might develop intracranial complications, but when the labyrinth became invaded then the risk of intracranial complications was greatly increased. Statistics varied, but undoubtedly the risk to life was very much greater. Consequently a knowledge of the pathological changes which occur in the labyrinth, and improved methods of investigating these changes were very important.

Dr J. S. Fraser, in reply, thanked the President for his kind remarks, and said that the paper was the result of seven years' more or less continuous work. He regretted that before beginning to speak he had forgotten to thank his chief, Dr Logan Turner, for his great kindness in providing him with the opportunity of doing this work. All along Dr Turner had given him great encouragement, and, but for him, it would have been practically impossible to do the work, because the vast majority of the cases were from Dr Turner's ward. When, in showing the slides, he used the words "my own case," he merely intended to convey the fact that the microscopic specimens were made by himself. He was also very greatly obliged to Dr Turner for the kind remarks he had made.

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2. NOTE ON A CASE OF SQUAMOUS EPITHELIOMA OF THE POST-CRICOID REGION, SECONDARILY INVOLVING THE LARYNX, TREATED BY RADIUM

By Dawson Turner, M.D., F.R.C.P., Medical Officer in charge Radium Treatment, Royal Infirmary, Edinburgh

A female, aged 38, recommended for radium treatment on 19th September 1913, by Dr Logan Turner. Duration of illness about six months. Patient has suffered from pain shooting up to the right ear, and from increasing dysphagia and progressive emaciation.

A gastrostomy was performed to relieve the dysphagia.

The right vocal cord was fixed. As the condition appeared to be inoperable, recourse was had to radium treatment as a palliative measure. Attempts made to apply the radium by the mouth failed. On 22nd September 1913 a tracheotomy was performed to relieve dyspnœa; afterwards a subhyoid pharyngotomy. Through this incision a tube of radium bromide (10 milligrams international standard) was introduced and pushed down into the growth. It was, however, found that the tube would not remain in the growth, mainly because the growth was very rapidly disintegrated by the radium; in the end the tube was allowed to remain on the top of the neoplasm. The tube was removed on 6th October. A considerable diminution had now occurred in the growth, and the patient's general health had improved. She was now sent home. On November 17th she returned. Only a stump of the growth could now be observed, and into this a tube of radium was again introduced. Again the tube would not stay in, and it had to be allowed to rest on the top. On December 4th the tube was removed, and the patient sent home. On January 24th she returned, having gained 3 lbs. in weight. Dr Logan Turner found that there was still some growth present on the right side. A tube of radium was again placed on the surface of the neoplasm, and withdrawn on 3rd February 1914. The total dose amounted to 5090 milligram hours. A considerable amount of slouching had been produced by the action of the radium. The patient was again sent home. The chief point of interest in this case was the remarkable action of the radium in dissolving away the growth, and in thus prolonging the patient's life. The symptoms were also relieved, and the patient gained in weight. Had it been possible to get all round the growth so as to attack it with the radium from every side, it appears probable that the whole of the neoplasm would have been removed. Only in sarcomas have I previously observed such a rapid process of disintegration. Other observers (Wickham and Degrais, etc.) have reported as to the beneficial effects of radium in cancer of the larynx, and the conclusion seems to be that in many such cases where operation is undesirable it might prove of service in relieving symptoms and prolonging life.

Discussion

The President said the Society had listened with much interest to Dr Dawson Turner's paper, and they were glad to know that

in radium there was a means of treating malignant cases in which other treatment failed. It was of interest to note the local effect of the radium, and also the benefit of small quantities applied over a long period of time.

Dr Logan Turner had hoped that Dr Dawson Turner would have brought some more cases before the Society: one could not draw deductions from a single case. The only deduction that could be drawn in this case was the prolongation of life. The patient was first seen in May 1913, and again in September, when the growth had increased considerably. Had that patient been left alone she would undoubtedly have been dead within a month or two. She was very anxious that her life should be prolonged, and when seen a week ago was in very fair health. She was unable to feed through her œsophagus. Undoubtedly the growth was still present, and gradually it would kill her, but she was very glad that her life had been prolonged, because she had three young children who were very much dependent on her. How far life was worth living with a gastrostomy, tracheotomy, and pharyngotomy was a matter of considerable question. An estimation of the duration of life in 21 of his cases of carcinoma in this region, after the patient was seen and the diagnosis made, shows that it varies from three to three and a-half months, some patients only living a few weeks, and other for six or seven months. This woman was still alive, and he should not be at all surprised if she lived for a considerable period.

Mr Scot Skirving thought the interesting point was that the case was one of *epithelioma*. Those who had had the benefit of Dr Dawson Turner's help in various cases would agree that in the majority of cases where manifest benefit had occurred or where apparent cure had resulted, the case has been one of sarcoma. When an inoperable patient happened to have an epithelioma, those who invoked the aid of radium did so with much less expectation of anything really valuable happening than in the case of sarcoma. He was entirely at one with Dr Logan Turner in wishing that they might have had a *group* of cases either of sarcoma or of epithelioma or of both. Nevertheless he was quite glad to have one case, because, when a similar case had to be dealt with, one could hold out some hope to the patient's friends, telling them that at any rate one knew of one case of epithelioma where life was prolonged. In one case of recurrent epithelioma, through the kindness of Dr Dawson Turner, he (Mr Scot Skirving) had been able

to give radium a really good trial. The patient was a man with a squamous epithelioma of the floor of the mouth, involving the lower jaw, slightly involving the tongue, and with pronounced secondary enlargement of the glands in the neck. When the patient was first seen, three years previously, a fairly extensive operation was performed—half of the lower jaw, half the tongue, and the whole glandular area on the affected side were removed. He (Mr Scot Skirving) had had the temerity to imagine that he had performed a radical cure when, eighteen months ago, the patient reported himself. Unfortunately, however, a month or two later he returned with a very evident recurrence—a recurrence which progressed with tremendous rapidity until a year ago he had a huge enlargement of the affected side of the face, with the cheek swollen, and a great deal of œdema of the forehead and eyelids. He was miserable, suffering some pain; the growth had fungated into his mouth, and it was a question whether to send him straight to the Longmore Hospital or to give him daily hypodermic injections of morphia. First of all, however, radium was tried, applied to the outside without much progress or improvement. Then tubes of radium were inserted into the affected parts for periods varying from six hours to four days at a time, through special incisions. After this the patient began to make progress, and very great improvement took place. A considerable amount of the growth sloughed away. The improvement in the patient, and especially the sense of well-being that resulted, was very remarkable. The man was not cured, far from it—he was now in the Longmore—but the growth had been reduced. Anyone who had seen the case at the beginning and at the end of the radium treatment would have had no hesitation in saying that the radium had not only prolonged life, but had reduced the growth to a large extent, and made the patient much more comfortable.

With regard to the different kinds of epithelioma, a distinction must be drawn. If a patient was reported to be cured who had had one of these very chronic epitheliomata of the breast, that would not, he thought, be very much help to the person anxious to advocate the claims of radium. On the other hand, if one took the case of a recurrent epithelioma of the tongue, where the progress of the case is notoriously very rapid, benefit from radium treatment was much more impressive. He thanked Dr Dawson Turner for his kindness in coming up at all hours, often at times inconvenient to himself, in order to change the radium tubes in the patient referred to.

Dr Dawson Turner, in reply, expressed his thanks to Dr Logan Turner and Mr Scot Skirving for giving him the opportunity of trying radium in these two cases. The kind of growth he had found most amenable to treatment by radium was that in which there were actively growing cells; the more fibrous, slow-growing growths were not so amenable. He had had several cases of scirrhus, in which the large hard growth flattened down and practically disappeared, but it was in sarcomas that radium was at its best. If Mr Struthers would take a look occasionally into the Radium Department, he (Dr Dawson Turner) would show him cases in which he might follow up for himself the benefit that was practically invariable after the use of radium. But the radium did not get a fair chance, for 99 cases out of 100 sent to him were hopeless, inoperable cases.

As to absolute cure—Did the knife produce an absolute cure? Sometimes. The knife may relieve the disease, but if it is in the patient's blood what could the knife do? As regards the cases of cure by radium which he had himself observed, there was a case of Professor Alexis Thomson's where the patient suffered from a recurring sarcoma in the neighbourhood of the clavicle. It was inoperable. Professor Alexis Thomson said at a meeting of this Society, and he should like to refer Mr Struthers to his (Dr Dawson Turner's) book which quoted that case, that the patient would certainly have been dead within three months had it not been for radium. In that case the result of the remedy applied externally and internally had been to remove the disease, because it was now more than two and a half years since the tumour disappeared under its action.

Meeting VII.—May 6, 1914

Dr JOHN PLAYFAIR, *President*, in the Chair.

I. EXHIBITION OF PATIENT

Dr Norman Walker showed—

Case of MYCOSIS FUNGOIDES.

II. Original Communications

1. EMPYEMA IN CHILDREN

By H. G. Melville Dunlop, M.D., F.R.C.P.,
Consulting Physician, Royal Hospital for Sick Children, Edinburgh

The following paper is based upon 98 cases of empyema which were treated in my ward in the Sick Children's Hospital, and the observations and conclusions arrived at are drawn from an analysis of these cases. As an indication of the frequency of empyema in children I would point out that during the same period in which these cases occurred, 861 cases of pneumonia were treated in the ward, being a proportion of one case of empyema to every eight or nine cases of pneumonia.

The pleural fluid effused during childhood appears to have a marked tendency to be of a purulent character, and the younger the child the more pronounced does this tendency become. Indeed it may almost be taken for granted when an effusion takes place into the pleura in a child under three years, that it will be of a purulent character. In 59 cases of pleural effusion in children under three years old the fluid was purulent in 53, and in the other 6 it was either serum or turbid serum. I believe in the majority of these cases in which turbid serum was found, if they had been left for a few days instead of being aspirated and cured, that they would have become purulent, and ought therefore to have been classed as empyema, instead of being entered in the case-books as serous pleurisy.

After three years the liability to the fluid being purulent gradually lessens till the age of ten, when the tendency to a serous effusion is established in the great majority of cases.

These facts are strikingly demonstrated in the annexed table of 149 cases of pleural effusion.

Under 6 months, 3 cases all purulent.										
Between 6 months and			1 year		9 cases,		8 purulent,		1 serous.	
,,	1 year and		2 years		26	,,	24	,,	2	,,
,,	2 years and		3	,,	21	,,	18	,,	3	,,
,,	3	,,	5	,,	23	,,	18	,,	5	,,
,,	5	,,	10	,,	57	,,	27	,,	30	,,
		Over	10	,,	10	,,	1	,,	9	,,

The fluid drawn off was invariably submitted to a bacteriological examination, and the results obtained are very similar to those of other observers.

The pneumococcus was present in pure culture in 53 per cent. of all the cases. When the pneumococcus is accountable for the effusion, the pus is opaque and greenish in colour, of a thick, creamy consistence, and generally associated with large masses of fibrin.

Next in order comes streptococcal infection, which was present in 16 per cent. In such cases the pus was much thinner in character and frequently on standing gave a whitish-grey deposit. In 14 per cent. the culture showed a mixed infection of streptococcus and pneumococcus. When this occurred the pus was often thin and watery at first, but became thicker later, owing to the pneumococcal infection. The staphylococcus alone was present in 3 per cent., and in two of the cases where it was found the pus had an offensive odour. The tubercle bacillus occurred in 3 per cent., while other forms and no growths occurred in 6 per cent. of the cases. The tubercle bacillus being difficult to find, I expect it was probably present in some of these cases where no growth was found. The fluid in the majority of these cases was of a turbid character.

These results are depicted in the accompanying table:—

In 53	per cent.	the pneumococcus	was	present alone.
,, 16	,,	,, streptococcus	,,	,,
,, 14	,,	,, pneumococcus and streptococcus	were	mixed.
,, 3	,,	,, staphylococcus	was	,,
,, 1	,,	,, staphylococcus and pneumococcus	were	,, mixed.
,, 2	,,	,, staphylococcus, streptococcus, and pneumococcus	,,	,, mixed.
,, 3	,,	,, tubercle bacillus	was	,, alone.
,, 6	,,	no growths.		

In determining the causation of empyema in children it is frequently stated that it occurs in two forms, viz. the primary and secondary, and that primary forms are by no means uncommon.

While admitting the occurrence of primary forms due either to cold or injury, I believe that in the majority of cases the condition is a secondary one and that primary cases are exceptional.

Of course, it may be argued that in hospital practice, with the exception of cases developing in the course of a pneumonia, the physician does not have an opportunity of seeing the commencement of the effusion, but we almost invariably got the history of some lung or other antecedent trouble. In only 7 per cent. of my cases did I fail to find some such history, and even in these the origin must remain in doubt, as there are so many channels through which infection may take place.

In the majority of cases the effusion either occurred with or followed lobar pneumonia, and when such was the case the fluid was invariably purulent. Empyema, according to my statistics, was much less frequently associated with broncho-pneumonia. It frequently developed subsequent to an attack of scarlet fever, measles, whooping-cough, and occasionally after influenza. Any suppurative process such as a purulent arthritis or an osteomyelitis may account for it, and in young infants septicæmia is a common cause. In striking contrast to empyema in the adult we only find a very small proportion in children due to tubercle. The causation in my cases are set forth in the annexed table :—

Lobar pneumonia	69 per cent.
Infectious diseases . . .	11 ,,
Broncho-pneumonia	5 ,,
Suppurative processes . . .	3 ,,
Tubercle	3 ,,
Influenza	2 ,,
Causes unknown	7 ,,

In most of the cases in which I had the opportunity of watching the development of the disease, the effusion occurred in cases of pneumonia within a few days of the crisis. Sometimes the symptoms heralding the onset of empyema were acute and violent, while in other cases the symptoms were more insidious and indefinite, and the illness developed gradually. I have never been quite able to grasp the significance of the cases described as latent empyema, unless the term is meant to apply to cases developing gradually. Some years ago I was greatly impressed on reading an article on empyema by a writer—his name has escaped me—that latency in empyema was synonymous with carelessness in the physician. With this remark I cordially agree, and I think that every physician attached to a children's hospital will confirm

the truth of this statement. Very frequently has it been my experience to have a child, generally a young infant, sent to me with the diagnosis of atrophy or miliary tuberculosis, and the general appearance went far to confirm such a diagnosis. We found the infant in the last stages of emaciation, virtually a bag of bones, with a dry, harsh skin, a peevish cry, and an appearance of intense exhaustion, but on making an examination of the chest we found it full of pus.

Probably the history is, that some months or weeks previous the child had been treated for an attack of pneumonia, which had passed off, but instead of making a satisfactory convalescence it had progressively wasted. The physician in attendance was probably quite able to diagnose empyema had he taken the trouble to examine the chest, but relying on his previous diagnosis, he had failed to do so, and thus fallen into error. The lesson to be learned is, that it ought to be a routine practice to examine, from time to time, every case we are attending which is not making satisfactory progress. I imagine there are few of us who at one time or another have not been guilty of a similar mistake. In six cases of young children an attack of convulsions was the earliest indication of the onset of empyema, while the other symptoms in the order of frequency were fever, cough, vomiting, quick breathing, sweating, restlessness, and delirium. When the disease attacked older children, and especially was this liable to occur after infectious diseases, the foregoing symptoms were less pronounced, but the child gradually assumed a languid appearance, became peevish, and had an anxious, careworn look, suffered from a short paroxysmal cough, and emaciated rapidly. In my series there were 53 boys and 45 girls. The left side was involved 45 times, while right-sided effusion occurred in 39 cases, which is not at all the usual proportion, or what we would expect from the frequency with which left side pneumonia occurs. Both sides were involved in 4 cases, which is, I think, about the general average. The amount of fluid varied greatly both in the quantity (from three-quarters of an ounce to 40 ounces), and in the time it took to gather. In some of my cases I found one side of the chest filled with pus from apex to base in the space of a few days, while in others the effusion took a very much longer time to collect. When a purulent effusion has taken place into the pleura, there are a number of symptoms which are very suggestive of the condition. First and foremost I would emphasise the statement, that in almost every case the child looks

seriously ill. It is surprising with what regularity succeeding residents have noted this fact in their case records. Whatever else they have omitted, they have almost invariably stated that "the child looks very ill." Not only does he look very ill but he has an anxious, pinched, almost frightened expression, and is profoundly anæmic. The pallor of the skin of the face is very marked, sometimes of an earthy colour, at other times of a straw-yellow tint, and often accompanied by some puffiness round the eye suggestive of Bright's disease. The exudation of pus into the pleura is almost invariably accompanied by very rapid and sometimes extreme emaciation. This symptom is seldom wanting, and I regard empyema as one of the most characteristic of the wasting diseases of children. When a child is sent to me as suffering from marasmus I always exclude these diseases, viz., tuberculosis, congenital syphilis, and lastly empyema, before coming to the conclusion that the diagnosis is correct. The muscles become flabby and wasted, and the child appears prostrate, exhausted, and suffering from extreme cachexia. There is usually some cough, either suppressed or paroxysmal in character, and the breathing is accelerated, but never to the same extent as we find in pneumonia. Indeed, it is surprising even when the pleura is full of pus how little real dyspnœa we find. There is generally a rise of temperature of from three to four degrees, and it may be very high, but on the other hand, especially in cases where the emaciation is marked and in young infants, there may be no rise of temperature at all, and it may even be subnormal, which is a very misleading sign. In ten of my cases there was either a normal or subnormal temperature during the whole period the child was under observation. When an empyema has lasted a long time without being operated on there is often clubbing of the fingers, and I have on one or two occasions witnessed these symptoms after a few weeks. A leucocytosis of from 20,000 to 30,000 is generally present when the fluid is purulent. Occasionally we find the cheeks flushed and the child perspiring, but more often the skin is pale, dry, and harsh. It is often mentioned as helpful in the diagnosis that we find diarrhœa, but my experience does not bear out this statement, and few of my cases suffered from this symptom. Such, then, are the symptoms characteristic of empyema; they form a very striking clinical picture. I shall refer very briefly to the physical signs, but there are one or two points I would like to emphasise, for though they have frequently been written about they will bear repetition. The

first point I would like to make is that whenever we find absolute dulness of a boardy character at the base, accompanied by a boxy percussion note at the apex, we should suspect a pleural effusion, especially if we get loud, harsh, exaggerated breathing above the region of dulness and on the opposite side. The sense of resistance communicated to the percussing fingers is another point of great value in children, but it requires some practice to appreciate the full significance of this sign.

I would further like to lay stress on the fact that it is quite common to get loud tubular breathing over a purulent effusion in the child. To those who are only familiar with the interpretation of physical signs in the adult this finding in the child is very misleading, but every one associated with a children's hospital knows how commonly either loud or distant tubular breathing is present in these cases. Vocal fremitus is of little help in children, the vibrations of the voice being of too rapid a character to be perceptible to the human hand. There is of course deficient expansion on the affected side, and in many of my cases there was marked flattening, due, I suppose, to collapse of the lung. Owing to the uncertainty of many of the physical signs, displacement of the heart's apex is a very valuable one in the diagnosis of fluid in the chest. This displacement is more marked in left-sided effusion, and when a considerable quantity of fluid is present we may find the apex beat in the epigastrium or even as far out as the right mammary line.

The complications of empyema are generally the result of further pneumococcal infection.

By far the commonest complication was purulent pericarditis, which was present in 30 per cent. of the fatal cases. It was most frequently associated with left-sided empyema in young infants. This condition was frequently only suspected, and as frequently altogether undiagnosed during life, as friction was seldom if ever detected, and the percussion area of cardiac dulness was little altered, owing to the small amount of pus present in the pericardium. Pericarditis should always be suspected after operation when the pleura is draining freely, if the child continues to look ill, with an anxious appearance, when dyspnœa is present, and the pulse continues rapid. Purulent meningitis and purulent peritonitis occurred several times, but I had no cases of other suppurative arthritis or general septicæmia.

The diagnosis both as to the presence of effusion and the nature

of the fluid is frequently difficult and sometimes impossible without the aid of an exploring needle, and fortunately the employment of this measure is attended with little risk, and whenever there is a patch of dulness which does not clear up it should be had recourse to.

When there is a reasonable certainty that pus is present the needle may have to be passed repeatedly before the fluid is struck, and in some cases even then it is of too thick a consistence to pass through the needle, while in other cases large masses of lymph block the lumen and prevent the passage of pus. The signs of the presence of fluid on which I place most reliance are displacement of the heart, dulness of a wooden character and accompanied by a sense of resistance on percussion at the base, and surmounted by an area in which we get a typmanitic note. The auscultatory signs are in my opinion of much less value than those obtained by percussion in determining the presence of fluid.

To discriminate between a purulent and serous fluid is often impossible, though there are many signs which favour the presence of pus rather than serum. It is frequently taught that a hectic temperature is indicative of pus, but I find as high temperatures recorded in my serous as in my purulent cases. When an effusion occurs in a child under three, the probability is strongly in favour of its being purulent, as also when it occurs subsequently to an attack of pneumonia, scarlet fever, or other infectious disease. Other symptoms in favour of pus are great emaciation, sallow tint, pallor of the skin, puffiness under the eyes, and a marked leucocytoses. The difficulty of differentiating an empyema from a pneumonia is immensely greater in the child than in the adult.

In the child we have no expectoration to help us, and, as already indicated, vocal fremitus is of little value. When to this is added the fact that we frequently have loud tubular breathing over an effusion, the value of an exploring syringe as a diagnostic help will be appreciated. The diagnosis between a chronic caseous pneumonia and an empyema, both of which give the same dulness and feeling of resistance on percussion, is frequently hopeless without exploration.

The prognosis of pleural effusion in children is rather difficult to estimate, as the mortality from the disease is largely determined by the cause and duration of the illness, and the age of the child.

In 98 cases I had 20 deaths, a mortality of 19 per cent. Under 2 years the prognosis is very grave, for in 30 cases I had 11 deaths,

a mortality of 36 per cent. At this age children seem to be specially liable to develop purulent pericarditis and meningitis, which are very fatal complications. After 2 years there is a very marked diminution in the mortality, and in 68 cases I had 9 deaths, being a death-rate of 13 per cent. When the effusion is double the outlook is most serious, for out of 4 cases only 1 recovered, but all these cases were in young infants. Pneumococcal cases over 2 years almost invariably recovered, whereas in streptococcal and mixed infections the prognosis was not nearly so good.

Thus out of 10 deaths over 2 years only 3 deaths occurred in pneumococcal cases, whereas the other 7 deaths were due to cases caused by other infections. Tubercular empyema is fortunately a rare occurrence in the child, and when it does take place the prognosis is not nearly so unfavourable as in the adult. Though the immediate outlook is not nearly so bad, the risks of a further tuberculous development must be borne in mind. The longer operation is deferred the worse the prognosis, as there is the added danger of the lung not expanding. The usual causes of death are bronchitis, broncho-pneumonia, pericarditis, peritonitis, and septicæmia.

It should be a cardinal rule that as soon as we recognise the presence of pus in the pleural cavity, it should be evacuated at the earliest opportunity. Success in treatment depends largely upon its early removal, and if we can secure good drainage and keep the cavity free from sepsis, the risks of complications occurring are greatly diminished. I believe that the earlier the evacuation of the chest takes place the less chance is there of the development of purulent pericarditis and meningitis, which I consider are in the main due to the long continuance of pus in the pleural cavity. In most cases any attempt to relieve the effusion by aspirators is a dangerous waste of time, and is by no means an efficient method of treatment, but there are exceptions to this rule. In cases where the exploring syringe has drawn off turbid serum—by which I mean serum charged with pus cells—I have frequently aspirated with excellent results. Then, again, it should be tried in very young infants who are unable to stand a serious operation. Aspiration is also useful when the effusion is very large. It may in such cases be had recourse to on the day previous to incision, so as to avoid the danger of syncope due to the sudden evacuation of a large quantity of fluid. In small localised collections of pus it is also recommended, but I have had no experience of such cases. The

objections to aspiration are that by this means we cannot remove all the pus, that large masses of fibrin are left ; we get no drainage, and have generally to resort to other measures later. As a rule our choice lies between resection of a portion of rib and simple incision of the pleura, and these are points in favour of each method. By the excision of a portion of rib we undoubtedly get better drainage and less risk of sepsis, but on the other hand it is a most serious operation, takes longer time, and causes greater shock. Incision of the pleura is a very simple and easily performed operation, is followed by little shock, and the drainage is usually sufficient. This method is specially indicated in young infants, and should the drainage prove defective or the discharge become offensive, resection can always be performed later. For many years it was my practice to have all my cases resected, but latterly I have had equally good results from simple incision.

8	cases	were	aspirated	with	1 death.
61	,,	,,	resected	,,	14 deaths.
29	,,	,,	incised	,,	5 deaths.

It should be an invariable rule to ascertain the presence of pus before operating by inserting an exploring needle even though pus may have been obtained on a previous occasion. The main danger of the operation is syncope and death from the anæsthetic, and it is safer in young children to trust to a local anæsthetic. I had one death on the operating table in a young infant. The main indications are to have a free incision and good drainage. The incision should be made in the sixth interspace in the mid-axillary line, and should be two or three inches long. When the chest is full of pus it is better, after incising the pleura, to insert the drainage tube at once and apply the dressings, by this means allowing the pus to escape gradually and thus avoiding a too rapid emptying of the pleura. In the majority of cases I believe the tube is left too long *in situ* and causes a discharging sinus. In many cases it can be removed in a week or ten days, and as a rule does not require to be retained longer than three or four weeks. In the cases which were resected the average time the tube remained in the wound was twenty-eight days, whereas when simple incision was practised the average time was thirty-six days.

Discussion

The President said the Society had listened with very great interest and pleasure to Dr Melville Dunlop's paper, and, he ventured to think, had derived a great deal of profit from it; they were much indebted to him for bringing the subject forward. In the few minutes at one's disposal it was impossible to allude to many of the points raised. With regard to the question of diagnosis, he quite agreed that percussion is far more valuable than auscultation—there is a something in the feeling of resistance in a child's chest where there is fluid. Treatment by incision he thought the most valuable, one ought if possible to avoid the more serious operations, such as resection, from which he had seen great shock produced; incision, in his experience, was all that was necessary.

Mr Caird referred to the negative evidence which sometimes followed on using the exploring needle, owing to its point entering masses of fibrin. He asked if Dr Melville Dunlop had, under such circumstances, employed the method of Dr Truby King, who recommended that the barrel of the syringe should contain a little sterile water. In the event of a negative result this clear fluid might be introduced, and on withdrawal was returned turbid or opalescent, thus demonstrating the pathological condition.

Dr John Thomson said he had listened with great interest to the paper, and one or two points had struck him. One was with regard to friction in pneumococcal pericarditis. Friction was very uncommon: he had never come across it at all, although he had tried in a number of cases where there was reason to suspect the presence of pericarditis. He did not know that there was any explanation of this. One point Dr Melville Dunlop had not alluded to directly was the importance of the child's general health in deciding the question whether a pleurisy in a child is to turn into an empyema or remain a serous pleurisy. In private practice, where the children are better nourished, empyema was a much less frequent occurrence, in his experience, than in hospital practice. Many years ago when in New York, and visiting Mount Sinai Hospital, Dr Schreiber, the physician, drew his attention to this fact in a very striking way. Mount Sinai Hospital is maintained by Jews, and a large proportion of the children are Jews from various parts of Europe. Dr

Scharlau pointed out that these Jews have to show a certain amount of money before being allowed to enter the States, and they collect it at the expense of themselves and their families ; the children are therefore in a bad state of nutrition. Any of these children are apt to get empyema if anything goes wrong with the chest. In one ward there were 16 children, and 8 of them had empyema. Then, he believed rather more in simple aspiration than Dr Melville Dunlop did. It depended on the state of the child : he was in favour of aspiration first in a great many of these cases. If the child was in a very low state, he did not know that it was worth while, but where the child has been fairly healthy before, he had seen two or three dozen cases where a single aspiration was sufficient to cure the patient.

Dr Goodall said on the billet Dr Melville Dunlop had stated " The misleading nature of physical signs as compared with adult interpretation." He would like that to read : " The misleading nature of the physical signs as compared with serous effusions." Two or three times he had seen cases of big empyemata where percussion dulness was marked, but where there was loud bronchial breathing. More than once in post-mortems he had seen cases where big empyemata had not been discovered or dealt with during life. Pathologists, if they cared to tell tales out of school, he was quite sure would agree that this was a fairly common experience. A month ago it had again been impressed upon him. A surgical colleague asked him to see a man who had been operated on twice for an obscure abdominal condition—an intestinal obstruction. Nothing had been found. There was marked dulness and resistance over the chest, but the bronchial breathing was so marked and loud it was agreed to temporise. Not feeling quite comfortable, he went to the ward the next day armed with a syringe in order to make a puncture if the surgeon agreed. He not only heard that the patient was dead, but that there was to be a post-mortem. A very big general thrombosis of the mesenteric vessels and empyema were found. The surgeon and he could not cast reflections at one another, but no doubt each thought a good deal !

Dr Cowan Guthrie brought forward a simple method which he thought worthy of the consideration of the Society, which might be adopted in cases of empyema, and could be performed under local anæsthesia. In a case of very severe pneumonia he injected

anti-pneumococcic serum. This helped the child considerably. As the symptoms recurred later, anti-pneumococcic serum was again injected. The pneumonia passed off, but was followed by extensive empyema or aspiration. Pus was found which, on examination, gave pneumococci in pure culture. As the pus once more refilled the pleura, St Clair Thomson's tonsil forceps were introduced into the tract left by the aspiratory needle. When the handles of the forceps were squeezed, an opening was made into the pleura, and the whole of the pus evacuated. Into the opening thus made the little finger was introduced, and then another finger still further dilating the opening, and finally two drainage tubes were introduced. The child made an excellent recovery.

Dr James Ritchie agreed with the previous speakers that they were much indebted to Dr Melville Dunlop. While the paper was being read the same thought occurred to him as to Dr John Thomson, namely, What was the health of those children who developed empyema? Because, as far as his experience went, empyema was not common in private practice. He would like to know what size of needle Dr Melville Dunlop used, whether large or small. Personally he always used a large needle with a perforation at the side, so that if anything happens at the point there is still the hole at the side. He agreed with Dr John Thomson that it was a wise thing to use aspiration first of all. Personally he would suggest that in young children aspiration should be tried twice. Pus in an adult should be at once removed by operation. The remarks as regards incision and resection had interested him greatly.

Dr Melville Dunlop, in reply, thanked the Society for the very attentive hearing, and the kind manner in which his paper had been received. If he had known of the Truby King process referred to by Professor Caird, it would have saved him a very great amount of anxiety in many cases. One case in particular he remembered, in which he was convinced there was pus in the child's chest. The exploring needle was passed nine times, but no pus was withdrawn; feeling certain, however, that pus was present, he asked Mr Stiles to operate; before doing so Mr Stiles passed a needle in five different places, but without securing pus. It was nevertheless agreed to operate. The pleura was opened, a piece of rib was resected, but still no pus made its appearance. The finger was then introduced into the thorax, and a large lump of fibrin extracted, which was followed by an enormous flow of pus.

He agreed with Dr John Thomson that the bearing of friction was a very unusual occurrence in purulent pericarditis in the child; and also that empyema occurred very seldom indeed in private practice. He was sorry, however, to disagree with both Dr John Thomson and Dr Ritchie that aspiration was an advisable operation in empyema, except in the instances referred to in his paper, viz., in very young children, and when there was a localised collection of pus. He had aspirated in a large number of cases of empyema, but was sorry to say that in the majority of these cases he had later to have the chest incised or resected.

With regard to Dr Goodall's remarks on auscultation, he had not sufficient knowledge of empyema in the adult to offer any criticism.

In reply to Dr Ritchie's question, he always used a needle with a large lumen, but even then often failed to get the pus, owing to blocking of the lumen.

2. OBSERVATIONS ON THE PATHOLOGY AND ETIOLOGY OF DUODENAL ULCER

By D. P. D. Wilkie, Ch.M., F.R.C.S.,
Assistant-Surgeon, Edinburgh Royal Infirmary

Ten years ago duodenal ulcer was regarded as a rare and obscure if somewhat dangerous malady, now we know it to be one which not only is of frequent occurrence, but can usually be diagnosed and successfully treated. The credit for this new knowledge belongs to surgery, and the names of W. J. Mayo, Moynihan, and Mayo Robson must always be associated with this advance.

The importance of studying the pathology of the living, which Moynihan has done so much to emphasise and illustrate, can hardly be over-estimated. At the same time it must be allowed that this study has its limitations as well as its great advantages, and that it may even, in the case of duodenal ulcer, be usefully supplemented by post-mortem room observations.

I have therefore little diffidence in bringing before you certain facts which, though supplemented by evidence from other sources, were gained mainly from the post-mortem room. During the past three years I have, through the courtesy of the pathologists of the Edinburgh Royal Infirmary, had the opportunity of examining

for duodenal ulcer and factors bearing thereon at some 400 post-mortems.* In this number I have met with 41 cases of duodenal ulcer. In only six cases had the duodenal condition any direct connection with the death of the patient—three deaths occurring after operation for perforated duodenal ulcer, one from a perforation not operated on, and two after gastro-enterostomy for a chronic duodenal ulcer. In only six cases had a diagnosis of duodenal ulcer been made before death, and all of these had been subjected to operation. At first sight this fact would seem to lend colour to the view that the diagnosis of duodenal ulcer is by no means so easy as many would have us believe. Whilst it is true that in several of these cases a large chronic ulcer with a marked stenosis of the duodenum was found, without there being any reference to digestive disturbance in a carefully taken case-record from the wards, it must be borne in mind that most of the patients had been under treatment for some other severe and eventually fatal malady, the symptoms of which overshadowed and obscured those of the duodenal lesion. At the same time, in one or two cases where a chronic ulcer was found in the duodenum, it could be confidently stated that it had caused during life none of the typical symptoms which we associate with the condition. There would appear to be a variety of ulcer, which might be termed "silent" duodenal ulcer, that may attain considerable size, and may even cause death by hæmorrhage or perforation, without producing any noteworthy symptoms. It was remarkable that the subjects of this "silent" type of duodenal ulcer were generally old people with arteriosclerosis, and in most, though not all, of them the ulcer was situated on the posterior wall of the duodenum. In the Edinburgh statistics[1] on perforated duodenal ulcer there were among the 200 cases recorded 17 in which no history of digestive trouble previous to the sudden perforation could be elicited. Gruber, from very extensive post mortem room observations, also found that 75 per cent. of the duodenal ulcers found after death had not been diagnosed during life. After making allowance for the special difficulties in obtaining a complete and accurate history in many of these cases, it was still clear from both these statistical studies that a chronic duodenal ulcer might exist without giving rise to the clinical picture which we regard as typical of this malady. Gruber[2] in his analysis, also noted the

* It should be stated that these represented barely one-half of the total post-mortems during that period.

frequency of symptomless duodenal ulcer in old people with arteriosclerosis. It must, I think, be allowed that the last word on the diagnosis of duodenal ulcer has not been spoken. The *syndrôme pylorique*, so clearly described by Soupault,[3] resembling as it does very closely the symptom-complex of duodenal ulcer as defined by Moynihan, may be and usually is associated with the presence of a duodenal ulcer, but not always is this the case. This *syndrôme pylorique* would appear to be the clinical expression of an irritable condition of the pyloric musculature favouring spasmodic contraction, a condition to my mind associated no less with the etiology than with the symptomatology of duodenal ulcer. The *syndrôme pylorique* may, however, be associated with ulcer on the gastric side of the pylorus, and there must be few surgeons who have not had the experience of opening the abdomen of a patient whose symptoms were those which are usually regarded as typical of duodenal ulcer, and of finding an ulcer in the lesser curvature of the stomach.

Morbid Anatomy

Site of Ulcers.—In all the 41 cases the lesion was situated in the first part of the duodenum, and with two exceptions within one inch of the pylorus. In regard to the exact situation of the ulcers in the first part of the duodenum, it was found that in 9 cases the ulcer was on the anterior wall, from half to three-quarters of an inch beyond the pylorus ; in 12 cases it was on the posterior, and in other 12 there was an ulcer on both anterior and posterior walls directly opposite—the "kissing ulcers" as described by Moynihan (Fig. 1). In 5 cases the ulcer was at the upper part or roof of the duodenum just beyond the pylorus, whilst in one case, a female subject, a large ulcer occupied the lower wall of the duodenum, extended on to the posterior wall and invaded the pylorus.

In 21 cases there was but one ulcer, in 16 two ulcers, and in 4 more than two ulcers.

These figures differ from those taken from operation records, in the relatively large number of ulcers on the posterior wall which they embrace, in the large proportion of cases with multiple ulcers, and most notably in the relative frequency of the "kissing" type of ulcer which they illustrate.

In some of the cases where an ulcer of fair size was found on

the posterior wall, when the duodenum was opened, no suspicion of its presence had been aroused by a careful examination of the unopened gut. The impression left by such cases is that this type of ulcer must be frequently overlooked at operation, and the question arises whether in doubtful cases the duodenum should not be opened and the mucous aspect of the posterior wall inspected.

In 5 cases of this series, besides the duodenal lesion, one or more gastric ulcers situated on the lesser curvature some distance from the pylorus were found.

Acute and Chronic Ulcer.—In regard to distinguishing acute from chronic duodenal ulcer, it is not so easy to speak with assurance as it is in the case of gastric ulcer. This is particularly true of ulcers situated on the anterior wall of the duodenum, as in many cases ulcers, which from the outside show evidences of chronicity, when viewed from the mucous aspect, closely resemble acute ulcers. Chronic ulcers on the posterior and postero-superior walls of the duodenum are usually more easily recognised, resembling, as they often do, the large crater-like ulcers found in the stomach. Thus although it was perfectly easy to say in some cases that the ulcer was of long standing, and in others that it was definitely acute, one hesitated in a certain number to venture an opinion as to the duration of the lesion. Mayo,[4] from an examination of ulcers excised from the anterior wall of the duodenum during life, was surprised to find how small and shallow were ulcers which from the outside gave all the appearance of chronicity. The following table indicates roughly the condition found among the 66 ulcers present in these 41 cases:—

TABLE I

Acute.	Chronic.	Doubtful Age	Healing or Healed.
16	20	11	15

Those in column 4 of Table I might perhaps with justice be classed among the acute variety, which is undoubtedly of frequent occurrence. The tendency of this variety to perforate has been duly emphasised by Grey Turner.[5]

ETIOLOGY OF DUODENAL ULCER

The essential factor for the formation of a duodenal as of a gastric ulcer is that a portion of the mucosa be so devitalised that

it may be digested and leave a breach of surface. For this initial devitalisation various causes have been assigned, *e.g.*—(1) the lodging of an embolus in an arteriole in the duodenal wall; (2) venous embolism, the embolus arising in the portal venous system and being carried in retrograde fashion up a duodenal vein; (3) toxic devitalisation of the mucosa, the result of some toxin circulating in the blood causing a degeneration, not only of the lining epithelium of the bowel, but of the endothelium of the capillary blood-vessels, so that small hæmorrhages take place and determine local areas of lowered resistance; (4) reflex nervous spasm of the vessels or of the muscularis mucosæ causing small hæmorrhages into the mucous membrane. I have sought carefully for evidence in support of these theories in 36 of the 41 cases which I examined.

Whilst arterial embolism may occasionally be the cause of an acute duodenal ulcer, there is no evidence that it is the usual mode of origin of such ulceration. In one case of my series where from endocarditis multiple emboli had been scattered throughout the arterial system, an acute ulcer in the first part of the duodenum, surrounded by a zone of venous engorgement, was apparently of embolic origin.

Von Eiselsberg first suggested that an embolus from a thrombosed omental vein might be carried in retrograde fashion up a gastric or duodenal vein and, lodging in the venous plexus of the mucosa or submucosa, cause localised infarction of the mucous membrane and thus an ulcer. Such a sequence of events has been shown experimentally to be possible,[6] and is a probable cause of some cases of sudden hæmatemesis occurring after an abdominal operation in which portions of omentum have been ligated. There is little or no evidence, however, to show that it is a usual cause of either gastric or duodenal ulcer. In spite of careful search I could find no facts in support of this theory in any of the cases in this series.

It is probable that a toxic degeneration or necrosis of the mucosa or of the lymph follicles of the duodenum, followed by an active digestion of the devitalised tissue, is the most frequent cause of acute ulceration. The source and nature of the toxin may vary widely; thus the products of tissue autolysis circulating in the blood cause degeneration in the parenchyma of various organs, among others the stomach and duodenum, and in the latter viscera the presence of digestive juices lends to digestion of the degene-

rated tissue and thus to ulceration. It is not surprising, therefore, that in patients dying some days after extensive burns gastric and duodenal ulcers should sometimes be found. In 2 of the 41 cases here dea't with, death resulted from widespread superficial burns, in the one case five, and in the other twelve days after the accident (Fig. 2). In each an acute duodenal ulcer of about 8 mm. diameter was found, and in the stomach great congestion, with superficial erosions of the mucosa. In the majority of cases, however, there was evidence of a primary cause within the abdomen, usually in the ileum, colon, or appendix.

In two instances where death resulted from acute intestinal obstruction (small intestine) an acute ulcer was found in the first part of the duodenum.

In four cases, all of chronic duodenal ulcer, there was definite evidence of stasis in the small intestine, i.e. the ileum was dilated and hypertrophied above an area involved in adhesions. In three of these cases, from the distribution of the adhesions which crippled the small intestine, it was obvious that the primary inflammatory focus had been in the appendix.

In four cases, three of chronic and one of acute ulcer, the patients had died from peritonitis resulting from acute appendicitis. In each instance there was a history of attacks of appendicular trouble previous to the final and fatal attack. In a fifth case, that of a woman who had died of mitral disease and bronchitis, an acutely inflamed appendix with localised peritonitis was found, and in the duodenum there were two large acute ulcers on the posterior wall. In a sixth case, where the patient died after operation for perforated duodenal ulcer, the distal end of the appendix was tense and club-shaped and contained pus.

In fifteen cases the most striking abnormality within the abdomen was the condition of the colon, and although a large number of different morbid conditions were represented in this group of cases, the one factor common to them all was faecal accumulation. In 11 cases there was an actual anatomical impediment at some part of the colon, in the remaining 4 there was merely great dilatation of the proximal part of the colon. In 3 of the latter the cæcum was prolapsed, dilated, and filled with scybalous faecal masses; in the fourth case, that of a woman whose bowels had acted only once a fortnight, the cæcum occupied the whole of the lower part of the abdomen, being filled with a great quantity of putty-like faecal matter, whilst the distal part of the colon was studded

with diverticula of varied size, most of them containing hard fæcal concretions.

Analysing the 11 cases in which a mechanical obstacle to the fæcal passage was present, we find that in 6 cases the obstruction was in the proximal part of the colon, and in 5 in the distal part. In all the former the impediment consisted in cicatrised pericolic bands constricting the ascending colon and having led to marked dilatation of the cæcum below. In one of them there was in addition a localised pericolic abscess, and a leak from this had led to fatal peritonitis. The pericolic bands here referred to were not merely the congenital parieto-colic folds so frequently met with where no evidence of stasis is present, but they were fibrous bands definitely constricting the gut. Whether they were the cause or the result of stasis is a matter on which opinions may differ. In the second group, where the obstruction was in the distal part of the colon, a variety of conditions was included. In one case there was an annular cancerous stricture of the pelvic colon of a very fibrous, scirrhous type, surrounded by numerous dense adhesions, and with great dilatation and hypertrophy of the colon above. In two cases a very pronounced degree of Payr's disease, or fusion of the distal half of the transverse colon to the descending colon, was present, and had led to great retention in the colon above. In one case the colon had not rotated and was lying on the left side of the abdomen, the small intestine occupying the right side. In this case the distal half of the colon was so matted in dense adhesions that the various parts of it could only with difficulty be identified. The cæcum was enormously dilated, and hid from view all the small intestine. In the fifth case the impediment resulted from a glueing of an unusually long pelvic colon to the mesentery of the ileum by dense fibrous adhesions. The fixation of the pelvic loop had evidently interfered with the passage of content through the colon, to judge from the accumulation of hardened fæces in the gut above. In two cases there was also pronounced cirrhosis of the liver.

In three cases there was no post-mortem evidence of trouble in the abdomen other than the chronic duodenal ulcer, and it is of interest that in each death was due to some form of cardio-vascular disease. From these observations I have become convinced that some definite relationship does exist between morbid conditions in the lower bowel and duodenal ulcer. From the varied character of the lesions found in the colon I think that it may be justly argued

that the duodenal is the secondary lesion, and this in spite of the fact that the constipation so regularly found in cases of duodenal ulcer frequently disappears after a gastro-enterostomy. What exactly the nature of the relationship between the two conditions is is not so clear. Whether it be that toxic absorption produces a reduction of the antipeptic content of the blood, as Katzenstein [7] suggested and Lieblein [8] has shown to exist in cases of gastric and duodenal ulcer, whether it be a primary action on the duodenal mucosa through the blood or only indirectly through damage to the liver, as the experiments of Gundermann [9] suggest, is uncertain. I am inclined to think, however, that it is in a combined action through the blood and through the autonomic nervous system that the connection is established.

Rössle,[10] who recently published the results of a similar investigation to that which I have been carrying out, was struck by the frequent association of chronic appendicitis and duodenal ulcer—16 times in 33 cases. He believes the connection between the two conditions to be through the nervous system, the irritation of the chronically inflamed appendix producing an irritable condition of the autonomic nervous system, expressed in the hypersecretion, the hypermotility, and the tendency to spasmodic contractions of the gastric and duodenal walls in the regions of the pylorus. The intensely interesting observations on the autonomic nervous system and the effect of drugs thereon recorded of recent years by Müller,[11] Eppinger and Hess,[12] Petrén and Theohari,[13] and others, and particularly the facts noted by Von Bergmann [14] regarding the irritability of this system and the increased vagotonus in cases of gastric and duodenal ulcer, are of importance in this connection, and there is some evidence to show that toxic absorption from the colon has a definitely vagotonic influence. The periodicity of the symptoms of duodenal ulcer, the effect of worry, over-fatigue, and of chills and vasomotor disturbances in determining the onset of a fresh attack all point to the importance of the nervous factor in duodenal ulcer. The irritable state of the autonomic nervous system induced and kept up by a chronic lesion in the appendix or colon readily explains those conditions of appendix and colon dyspepsia, which in many cases are the forerunners of gastric and duodenal ulcer. Whether we must postulate an initial toxic necrosis of the mucosa or its lymph follicles, or whether, as Boycott [15] and Resch maintain, hæmorrhagic erosions may occur and develop into ulcers merely from reflex spasmodic contractions of the muscu-

laris mucosæ, is a matter more of scientific than of practical interest. The important clinical point is, that from morbid conditions in the lower bowel functional and actual organic changes in the gastro-duodenal area may result, and that treatment, whether medical or surgical, must be directed to first causes as well as to secondary results.

Site of Duodenal Ulcer

The most striking feature of duodenal ulcer is the regularity with which it is found in certain well-defined positions. The characteristic positions on the anterior and posterior walls half an inch beyond the pylorus claim probably 85 per cent. of all duodenal ulcers. The roof of the duodenum just beyond the pylorus is the other common site, whereas the remainder of the first part of the duodenum, although frequently the seat of scars of healed acute ulcers, is seldom found involved by a chronic ulcer. Several facts may be mentioned which have a bearing on this curiously selective character of duodenal ulceration.

Embryologically, the first part of the duodenum resembles the stomach in that it arises from the foregut. Its mucous lining differs from that of the rest of the duodenum in being practically devoid of folds. Further, this part of the duodenum is rich in lymph follicles, and there is some evidence that many acute duodenal ulcers originate in the breaking down of such follicles. This fact was borne in on me forcibly by the case of a boy, aged 11, who died under the anæsthetic during an operation for intestinal obstruction. A coil of small intestine had been loosely snared by an omental adhesion to an old inflamed appendix. At the post-mortem it was found that the boy had a very pronounced degree of status lymphaticus. The lymph follicles in the pyloric end of the stomach and first part of the duodenum stood out prominently, and on the anterior wall of the duodenum, half an inch beyond the pylorus, was an ulcer of some duration, whilst on the posterior wall just opposite was the scar of a healed ulcer. In acute infections, such as appendicitis, which have ended fatally, even though there be no duodenal ulcer, microscopic examination of the wall of the first part of the duodenum frequently shows breaking down of the central parts of the lymph follicles (see Figs. 3 and 4).

Exposure to Acid Content.—Radioscopy has shown that when food is taken into the stomach, almost immediately some of it

is passed on into the first part of the duodenum, and that this onward passage is unusually rapid in cases of duodenal ulcer. On entering the roomy duodenal vestibule there is a slight pause in the onward course, and this gives the well-known appearance of the "duodenal cap" first described by Cole.[16] In cases of duodenal ulcer this delay in the first part of the duodenum is more marked than normal, suggesting that there is some obstruction to the normal process of feeding of the more distal duodenum from the loaded vestibule, which Cole regards as a reservoir. Schmieden[17] and Dunkeloh have laid great stress on this retention of acid chyme from the stomach in the first part of the duodenum in regard to the etiology of chronic duodenal ulcer. Obstruction at the first duodenal angle is, according to Quimby,[18] a very common cause of gastric dilatation: it would certainly seem to be a contributing cause of duodenal ulcer. Kreuzfuchs[19] has shown that in cases of uncomplicated duodenal ulcer, whilst there is almost invariably hypermotility of the stomach, with rapid emptying of the organ in the first hour or two after a meal, a certain residue of what appears to be the coarser part of the food remains for a longer period in the stomach. The initial period of active peristalsis and open pylorus is succeeded by a period of pyloric spasm with the escape of but small quantities of a hyperacid content into the duodenum. This is the period of late or hunger-pain so characteristically relieved by alkalies and belladona. The primary stasis of a moderately acid content and the later arrival of small quantities of a hyperacid chyme are important factors in determining ulceration in the duodenal vestibule should any devitalised area of its mucosa be present.

Moynihan[20] expresses the opinion that the constancy with which chronic duodenal ulcer is found on the anterior wall just beyond the pylorus is probably to be explained by the impinging of the acid chyme on this spot as it is ejected from the stomach. The ulcer frequently found on the posterior wall just opposite the anterior one he regards as a "kissing" or contact ulcer. In regard to the first of these explanations, it is based on the view that "an enormous preponderance of ulcers occur on the anterior wall." Post-mortem evidence does not confirm this view, which is based on the examination of the duodenum from its external aspect. In regard to the so-called "kissing ulcers," I think it is very doubtful that they are contact ulcers. The active secretion which occurs in the duodenum, together with the fact that gas is very often

present as well, make it unlikely that the anterior and posterior walls can for any length of time be sufficiently closely in contact to permit of direct infection.

Blood-Supply of First Part of Duodenum.—In a former paper I [21] suggested that the blood-supply of this area of the duodenum might be responsible for the characteristic distribution of chronic ulcers. The situations of both the anterior and posterior ulcers correspond closely to the proximal boundaries of the areas supplied by the anterior and posterior branches of the supraduodenal artery. The anastomosis of the branches of this vessel with surrounding vessels, and especially with pyloric arterioles, is of the most sparing description. Any factor, such as arteriosclerosis, tension on the vessel, or spasm of the muscular coats of the bowel, which interferes with this vascular unit, will favour ulceration at the periphery of the area supplied by it, and in the subjects of duodenal ulcer the presence of such a factor is, I hope to show, not mere theory. Further, the local endarteritis of the terminal branches which results from ulceration in the duodenal wall must certainly tend to delay healing and to favour recurrence when healing does occur. The hyperæmia of the peritoneal coat over a duodenal ulcer so constantly seen at operations after the duodenum has been handled or exposed to the air for some minutes cannot be taken as an index of the blood-supply of the whole thickness of the duodenal wall at this area under normal conditions with the patient in the erect attitude.

Sex Incidence of Duodenal Ulcer

The relative frequency of duodenal ulcer in the male as compared with the female sex—in the operation statistics of Mayo and Moynihan it was approximately 4 to 1—was upheld in my most-mortem cases—33 males to 8 females. Such a constant ratio must depend on some constant and therefore probably structural difference in the two sexes. Such a difference is to be found in the form and position of the stomach and duodenum. In the female the stomach is longer in its vertical axis, the pylorus is on a lower level, and the first part of the duodenum is longer and laxer than in the male (Figs. 5 and 6). Though notable exceptions will be found to this sex difference in the shape and position of the stomach, I have satisfied myself from the examination of several hundred bodies that it is sufficiently constant to justify

the recognition of a male and a female type. This distinction, though not usually recognised by anatomists, is well known to surgeons, and makes the operation of pylorectomy a relatively simple operation in a female patient.

In a healthy subject whose abdominal muscles are in good tone the stomach, full or empty, is buoyed up by the intestines, and its supporting ligaments bear little, if any, strain. When, however, the tone of the abdominal muscles is either temporarily or permanently impaired, the viscera tend to sag downwards, and strain may be thrown on vascular supporting ligaments. The stomach is slung up in the abdomen by the two borders of the gastro-hepatic omentum, the left one containing the coronary artery, the right one, or hepato-duodenal ligament, usually containing the supraduodenal artery. The thin intermediate portion of the gastro-hepatic omentum can under no circumstances give support to the stomach.

Dunkeloh, in his X-ray observations on cases of duodenal ulcer, noted that a slight degree of gastroptosis was one of the constant features in such cases, a fact which Bier [22] has previously noted. Tension on vessel-containing ligaments usually leads to slight anæmia, and anæmia to muscular spasm in the wall of the hollow viscus supplied by the vessels. The anæmic spot on the duodenum which Mayo [23] observed at operations when this viscus was pulled out into the abdominal wound is an excellent illustration of this point. This sequence of events, i.e. tension on vessels, slight anæmia, and muscular spasm, will be particularly liable to occur in the stomach or duodenum of one who is the subject of hypervagotonus, and according to Von Bergmann, patients who suffer from gastric and duodenal ulcer invariably show increased vagotonus. If, then, we grant that strain, however slight on the supporting ligaments of the stomach and duodenum, may occur, it can be very readily demonstrated that this strain will be distributed between the two ligaments according to the relative height of the two ends of the weighted viscus. When the pylorus is low and the duodenum long and lax, as in the female type, almost all the strain is borne by the cardiac ligament containing the coronary vessels, whereas where the pylorus is situated relatively high up, where the first part of the duodenum is short and more or less fixed, a considerable amount of the strain is borne by the hepato-duodenal ligament containing the supraduodenal vessels (Figs. 5 and 6). On these lines one may explain why ulcer on the lesser curvature of the stomach is relatively common in women

and ulcer in the first part of the duodenum relatively frequent in men. It may also explain why rest in bed is so essential for the effective medical treatment of both forms of ulceration. It may be argued against this explanation of the sex incidence of duodenal ulcer that not so very rarely a duodenal ulcer is found in a female with visceroptosis and an exaggerated feminine type of duodenum. I have operated on two such cases, but in both, although the pylorus was very low (level of umbilicus), the greater curvature of the stomach had sagged down very much lower, so that really one had to deal with what was, mechanically, almost a male type of stomach and duodenum. Moreover, it is not altogether rare to find in the same individual an ulcer in both those situations which are exposed to strain, namely, the lesser curvature of the stomach and the duodenum just beyond the pylorus.

Conclusions

1. Duodenal ulcer is a malady of frequent occurrence and one which often passes unrecognised.

2. Although as a rule readily diagnosed, a chronic duodenal ulcer may occasionally exist and give rise to none of the characteristic symptoms, the first evidence of such a "silent" ulcer being sometimes its perforation.

3. "Silent" duodenal ulcers are met with most frequently in the subjects of arteriosclerosis, and are found for the most part on the posterior wall of the duodenum.

4. Some toxic or irritative factor, usually within the abdomen and most frequently associated with the colon or appendix, is found in a large proportion of cases of chronic duodenal ulcer.

5. Probably many acute duodenal ulcers are primarily follicular ulcers from the breaking down of inflamed lymph follicles.

6. Whatever be the primary cause of a gastric or duodenal ulcer, spasm of the muscular coats of the viscus is an important factor in determining its chronicity.

7. The situation of the opposing ulcers on the anterior and posterior walls of the duodenum on the boundary zone of the areas supplied by the anterior and posterior branches of the supraduodenal artery suggests that a common vascular deficiency rather than a contact infection accounts for the peculiar tendency to chronicity and recurrence.

8. This vascular deficiency may be due to arteriosclerosis, but

probably it is usually due to spasm of the muscular coats of the duodenum induced by a slight local anæmia consequent to strain on the supraduodenal vessels, this muscular spasm being favoured by the increased vagotonus and the irritable condition of the autonomic nervous system which exists in such cases.

9. The sex incidence of duodenal ulcer is to be explained on anatomical grounds. The relatively high pylorus and short fixed duodenum of the male allows of its vascular supporting ligament, the hepato-duodenal ligament being exposed to strain, which in the female, with her relatively low pylorus and lax duodenum, is borne by the left border of the gastro-hepatic omentum and lesser curvature of the stomach.

10. The fixity of the male duodenum further predisposes to kinking at the first duodenal angle and thus to an unduly long exposure of its first part to the acid chyme from the stomach, undiluted by bile or pancreatic juice, the regurgitation of which is impeded.

Discussion

The President said they had had a most interesting, instructive, and able paper from Mr Wilkie, and were extremely indebted to him for presenting the subject in such an able manner. He had made valuable suggestions as to the etiology. This was an ailment that had given all medical men a great deal of anxiety and trouble, it was therefore a great pleasure to hear suggestions made, and being a subject of general interest would no doubt give rise to a good discussion.

Mr Caird thought Mr Wilkie's lucid and suggestive paper had illuminated a difficult subject. He was specially interested in the remarks as to the relative frequency of duodenal and gastric ulcers from the sex point of view, and the anatomical considerations as determining this.

Dr William Russell said they were all, of course, impressed with the great excellence of the demonstration, he had been particularly interested, and would take up one or two points which seemed to him to be the most important. Firstly, the connection which had been suggested between conditions in the lower part of the alimentary tract and duodenal ulcer. He could support that proposition

in a somewhat interesting fashion. A great many cases diagnosed as gastric ulcer with a history of hæmatemesis were sent into his ward by able practitioners—he was not prepared to give figures, although no doubt he had them in his records. A large number of those cases in which one doubted, after careful consideration of the symptoms, whether the patients really suffered from gastric ulcer, got rapidly well under colon lavage and light diet, and were discharged with complete disappearance of all gastric phenomena. So much was this the case that now these patients were not put on diet suitable for gastric ulcer but were treated by treating the colon. He did not wish it to be inferred that there were no cases of gastric ulcer to be dealt with, but he was giving his experience in the Royal Infirmary during recent years.

The second point was the elaboration of the contention that acute duodenal ulcer, as acute gastric ulcer, was the result of local anæmia. He was glad to note that Mr Wilkie did not champion the old doctrine of embolism, and had abandoned the view, which he himself had suggested, of a backwash in the veins; that he argued in favour of the view that the local anæmia was due possibly to more than one cause, that there was local spasm and also different anatomical relationships, which determined the local anæmia in one sex more than in another. He (Dr Russell) had made and published the suggestion that local spasm was probably the cause of the anæmia which preceded the acute erosion; and the experimental work which had been done on the stomach and duodenum with adrenalin seemed very strongly to bear out that view. Further, it had to be remembered that both duodenal and gastric ulcer were definitely associated with hyperchlorhydria, that hyperacidity was the precursor unquestionably of the majority of cases of gastric and duodenal ulcer, and that the pyloric spasm which was associated so definitely with hyperchlorhydria was also the explanation of the localised spasm which went on to erosion. Then Mr Wilkie had obtained post-mortem statistics which were particularly interesting in view of the statement made not many years ago that one never saw duodenal ulcer or scar in the post-mortem room; Mr Wilkie had found about 10 per cent. of duodenal ulcers in the four hundred odd cases he had examined, showing that evidence of duodenal ulcer was fairly common in the post-mortem room. That it occurred as a silent ulcer, no one who seriously investigated those matters could have any doubt, and he had that day seen a patient who illustrated the point. The patient had been seen a year ago, after returning

from Australia. When leaving Australia he had a very severe melæna which lasted for some days and from which he nearly died; he recovered, picked up on the voyage, and had no recurrence of the melæna until a few days ago. In the interval there had been no symptoms referable to the duodenum, he had put on weight and improved in general health, and yet ten days or a fortnight ago he had another severe attack of hæmatemesis in London. Undoubtedly that patient had a silent duodenal ulcer which would most certainly have to be dealt with, and he should recommend it being dealt with without further delay. At the same time he did not think physicians, at all events, could take up the position that duodenal ulcer was to be diagnosed by the pyloric syndrome referred to by Mr Wilkie. It was very difficult to determine the presence of duodenal ulcer when dealing with hyperchlorhydria: personally he never diagnosed it unless in addition there was definite or "occult" blood in the stools. In conclusion he expressed his very great appreciation of the beauty and skill with which the communication had been made.

Professor Alexis Thomson thought they should restrict themselves, as far as possible, to the causation of duodenal ulcer, because that was the important part of Mr Wilkie's admirable paper. Mr Wilkie had perhaps laid too much stress on the pathology of the dead. One must be chary of accepting the view of the causation of duodenal ulcer by these bands described as supporting the stomach, and which had been emphasised as occurring in greater frequency in the male. One must also be chary of accepting the view that stasis in the lower part of the colon was the cause of duodenal ulcer, although it had the support of Dr Russell with his lavage, and the still more important support of Arbuthnot Lane. He could imagine no one listening with more satisfaction to Mr Wilkie's paper than Arbuthnot Lane, who holds that both gastric and duodenal ulcer arise from stasis, and that modern methods of treatment, notably gastro-enterostomy, are erroneous. It was not Professor Thomson's experience that duodenal ulcer was found in persons who presented the lesions figured on the screen, especially those in the lower part of the colon and contraction bands in the first part of the duodenum. Probably there is usually a combination of factors, and one must associate as a causal factor the simultaneous increase of duodenal ulcer with the increase of lesions of the appendix, probably the toxic factor which caused lesions of the appendix

had a great deal to do with the production of ulcer of the duodenum.

Stasis of the lower part of the alimentary tract seemed in direct opposition to the sex frequency. Duodenal ulcer is four times more frequent in the male than the female, yet the stasis referred to is, of course, very much more common and more pronounced in the female. In his experience, duodenal ulcer had occurred especially in comparatively young men in whom there had been no suspicion of any kind of stasis.

Mr Wilkie had established the fact that duodenal ulcer may occur as a result of burns, so that bogey had at last been laid at rest. Notwithstanding his criticisms, he had the heartiest admiration for Mr Wilkie's paper.

Dr Chalmers Watson joined with the previous speakers in expressing his appreciation of Mr Wilkie's extremely instructive paper ; the pathological researches and clinical examinations added greatly to its value. Assuming the correctness, as he did, of Mr Wilkie's contention that duodenal ulcer is secondary to a lesion lower down, the question naturally arises, Could that lesion have been the cause of death ?

Radiography was of very great value in regard to stasis. In the *Lancet* of May 2nd, Dr Cole had given a very interesting demonstration of duodenal ulcer obtained by radiography. Unless the examination is made in the recumbent posture it is not complete. He (Dr Chalmers Watson) divided cases into three classes, namely : (1) those in which an accumulation of bismuth in the site of the ulcer is demonstrated ; (2) those in which there is an irregular contraction of the duodenum, often associated with reverse peristalsis ; and (3) those in which definite retention of the bismuth can be seen in the duodenum many hours afterwards. He doubted whether the duodenal abnormalities shown by the X-rays are dependent on the disease in the duodenum or on the disease lower down, which Mr Wilkie had so well brought out.

He agreed as to the difficulty in diagnosis. In a case of obstructive jaundice which had been under the speaker's care, no adequate cause could be found at the operation, notwithstanding the fact that the whole region of the duodenum was explored. The patient, however, died a few days later, and post-mortem examination revealed a large ulcer of the duodenum which was undergoing malignant degeneration.

Mr J. W. Dowden remarked that an interesting point in the etiology was the large majority of the ulcers, whether on the anterior or posterior wall of the stomach or in the pyloric region, and that those situated nearer the cardiac end were also as a rule on the lesser curvature. In two cases of hairball in the stomach on which he had operated, one in an adult and the other in a child, the collection of hair was seen and felt, pressed firmly up against the lesser curvature of the stomach, and more densely packed at the pyloric region. The stomach itself was flaccid, and the lax walls lay quite free at the lower part. The impression produced by this condition was that traumatism played an important part in the formation of ulcer.

With the increase of appendicitis, gastric and duodenal ulcer had also increased. There were, however, a number of cases of dyspepsia associated with chronic appendicitis. Some of these cases even had symptoms of melæna and hæmatemesis cured by the removal of the appendix: in many cases of appendix dyspepsia there was hyperacidity, and in one, cured by the removal of the appendix, the patient was in the habit of taking four teaspoonfuls of bicarb. of soda daily.

Dr Cowan Guthrie said, after hearing Mr Wilkie's very excellent paper, one important method of investigation occurred to him, viz., the experimental method. By experiment one could determine whether a supposed cause of a disease was the true one or not. It was important in this connection to consider the function of the large intestine as compared with the function of the small: the large intestine acting chiefly as a drain, while the small intestine's function was chiefly nutritive. Anything which interfered with the proper action of the ileo-cæcal valve and allowed the passing back of the contents of the large into the small intestine must of necessity affect not only the appendix but also the small intestine, including the duodenum, and the specific organism which produces these ulcers is the communis coli, and the part affected depends upon the local condition of the bowel at the time. If in treating appendicitis by special method, to cover results, and particularly if in addition to the duodenal ulcer present which also gives way to the same form of treatment, it shows that practically all these cases are due to the same causative agency, viz. the communis coli. Recently he had had as many as twenty cases of appendicitis in which cure had resulted from antiseptic serum followed by autogenic vaccine. In one case in which the vaccine was not used, the condition recurred and

had to be operated upon. The serum alone was not sufficient to prevent a recurrence. In three other cases with symptoms pointing to duodenal ulcer, the injection of anticoli serum gave good results. His experimental experience led him to believe that duodenal ulcer is really due to conditions causing interference with the function of ileo-cæcal valve and therefore permitting the passage upwards into the small intestine of the contents of the large bowel, teeming with organisms of which the communis coli is the all-important one.

Mr Wilkie in reply thanked the Society for the very kind way in which they had received his paper.

Prof. Alexis Thomson had raised the very interesting point—Why, if constipation is so very common in the female, is duodenal ulcer so very common in the male? In the latter part of his paper he showed that the tendency in females was towards gastric ulcer, and he thought most surgeons would agree that gastric ulcer was very much more common in the female. Constipation predisposed to gastric ulcer in the female and duodenal ulcer in the male. He did not wish anyone to carry away the impression that everyone who was suffering from constipation would get a duodenal or gastric ulcer, but merely stated the fact that in a large proportion of cases examined post-mortem there was marked evidence of stasis in the colon.

Referring to Mr Struthers' remarks, he (Mr Wilkie) believed that the amount of strain necessary to produce a spasm was exceedingly slight. The least strain in a person with a hypertonic condition of the vagus, and therefore liable to spasm, might bring on spasm in that region. The cases met with post-mortem were on the whole older—most of them over forty and many over forty-five—than those met with in surgical practice. Those who died of appendicitis were, however, young people, below twenty-five.

In connection with Dr Russell's remarks on colon and appendix dyspepsia the prevalent view amongst German pathologists is that there is an irritation of the sympathetic in the lower part of the abdomen, the stimulation being reflected down the vagus, this stimulation of the vagus causing hypermotility, hypersecretion, and a tendency to spasm, just the conditions met with in appendix dyspepsia and duodenal ulcer.

Mr Dowden's remarks on hair-ball and pressure on the lesser curvature were quite in accord with his (Mr Wilkie's) views.

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Meeting VIII.—May 20, 1914

Dr John Playfair *President in the Chair*

This was a Clinical Meeting held in the Royal Infirmary.

I. Exhibition of Cases

1. *Dr Dawson Turner* showed—

A RECURRENT CARCINOMA in a woman, aged 57, recommended by Mr Miles. Right breast removed for a sarcoma in August 1912. Recurrence in October 1912. On admission on February 3rd, 1914, there was a large swelling measuring 3 by 2½ inches, and projecting 2½ inches from the sternum. Considerable pain. Treated by the internal and external application of radium. Total dose 5510 mg.-hours. Result, relief of pain and gradual disappearance of growth.

2. *Mr John Fraser* showed—

A child, E. C., aged 5 years. The child developed TUBERCULOUS DISEASE OF THE LEFT MIDDLE EAR AND MASTOID, with secondary involvement of the facial nerve, and resulting facial paralysis. Operative treatment of the ear condition failed to relieve the paralysis, and at the end of a year there was no sign of recovery.

The cervical glands became tuberculous, and 18 months ago they were removed. During the dissection the peripheral facial nerve was anastomosed to the central spinal-accessory: a portion of the external jugular vein was excised and used to encircle the line of suture. Now, at the end of 18 months, muscular power is returning to the face.

3. *Mr Alexis Thomson* showed—

(*a*) EXTRADURAL SUPPURATION following head injury in a man æt. 32; Pott's puffy tumour with signs of pressure on motor area; extensive removal of bone and administration of autogenous staphylococcal vaccine. Recovery.

(*b*) RESECTION OF HUMERUS FOR SARCOMA. The patient, a boy æt. 11, was shown to the Society in 1910, after an operation in September 1909, in which the middle portion of the humerus was resected for a spindle-celled periosteal sarcoma. The disease returned, and the lower half of the humerus was removed in July 1910, and again a portion of fibula was inserted in its place. The disease has not returned, and the humerus has grown in length.

4. *Dr Robert A. Fleming* showed—

(*a*) W. C., male æt. 18, suffering from CARDIAC HYPERTROPHY AND DILATATION, with remarkable præcordial pulsation.

Present illness believed to have started when a child of 7, but no very definite history of rheumatic fever.

Pulse 116, typical water-hammer. Has aortic stenosis with thrill and incompetence, and also mitral stenosis and incompetence.

(*b*) J. J., female, æt. 22, a case of GLYCOSURIA, presenting some remarkable features. Sugar in the urine has varied from about 400 to 80 grs. in the day.

I am indebted to Dr Cramer for the following notes of her urine examination: "Urine gave typical glucosazone crystals, fermented, rotated, polarised light to the right, and gave a positive Nylander reaction, but when tested with Fehling's solution gave scarlet red, which darkened rapidly and turned black, depositing a black precipitate. This abnormal reduction is due to production of a colloidal solution of cuprous oxide (red), which is further reduced to colloidal copper (black). The reaction is due to the presence of glucose in a relatively concentrated urine, which contains a high percentage of creatine and creatinine. If the urine

is diluted with water, the ordinary typical Fehling reaction is obtained.

"The occurrence of the reaction indicates a glycosuria which is not associated with a polyuria."

5. *Mr David Wallace* showed—

(*a*) CARCINOMA OF HEPATIC FLEXURE. Short circuit. R. H., æt. 55, was seized with sudden pain in right iliac fossa in April 1913. Previously he had had much constipation, and when he took medicine had much pain in the bowels. He gave a history of this for 12 months. A mass was felt in the right lumbar region, but there was no visible peristalsis. Operation 14th May 1913. Carcinoma of hepatic flexure, with secondary nodules in liver. Ileum laterally joined to transverse colon. Recovery. Now going downhill.

(*b*) RENO-PELVIC CALCULUS. H. W., male, aged 28. Pain in right lumbar region and blood in urine. X-ray showed stone in pelvis of kidney. Pain began in May 1912. Hæmaturia at intervals since then. Diagnosis not difficult. Operation 3rd February 1914. Stone removed through pelvis. Incision in pelvis closed, and line of incision strengthened with fascia. No escape of urine. Calculus similar to three others in having large acicular crystals on surface.

(*c*) Mrs C., RENAL CALCULUS in substance of kidney. Similar in appearance to that of H. W. Stone fixed in kidney. Symptoms anomalous, but X-ray gave definite shadow. Percussion sign present.

6. *Mr D. P. D. Wilkie* showed—

(*a*) Resection of posterior nerve roots for GASTRIC CRISIS OF TABES. Patient suffered from severe gastric crises, lasting last few for two days every week with severe pain and vomiting. The other manifestations of the disease were relatively slight. Operation: spines and lamina of third to eighth dorsal vertebræ inclusive removed. Seven posterior roots (5th to 11th) on each side resected. No pain or vomiting since operation.

(*b*) CONGENITAL ABNORMALITY OF COLON. Girl admitted to hospital suffering from peritonitis. Incision for appendix revealed only small intestine on right side of abdomen. Colon had not rotated and was lying entirely on left side of midline. Radiograph.

7. *Dr Chalmers Watson* showed—

Case of CHRONIC INDIGESTION. Young man aged 20, complaining of abdominal pain, nausea, flatulence, constipation, loss of weight, and general lack of vigour, associated with marked ileal stasis and secondary gastric and colon stasis, the primary factor being, in all probability, adhesions in the ileo-cæcal region. Transparencies of X-ray plates (Dr Hope Fowler) shown.

8. *Dr W. T. Ritchie and Mr J. M. Graham* showed—

Case of PERNICIOUS ANÆMIA treated by direct transfusion of blood. Mrs T., æt. 51; two previous relapses after arsenic treatment; red cells=770,000, Hb.=35 %; anæmia increased in spite of arsenic; transfusion on 8th March 1914, radial artery to cephalic vein—donor, daughter; thereafter numerous nucleated red cells appeared and condition steadily improved. 13th May—red cells =2,500,000, Hb.=65 %.

9. *Mr J. W. Dowden* showed—

(*a*) Man, æt. 29, showing power of locomotion in spite of absence of head and neck of femur. Charcot's disease of hip-joint without nervous symptoms.

(*b*) Woman, æt. 50, showing result of bone graft from crest of tibia implanted for un-united fracture of neck of femur, with absorption.

(*c*) Man, æt. 26, showing result of plastic operation for stiffness following on injury of finger.

10. *Mr Caird* showed—

(*a*) H. H., æt. 31, pitched from cycle against a wall; cycled 7 miles afterwards. Dressed by doctor. Two days later came into hospital. Operation; depressed fracture found. Bone elevated and pieces reimplanted; stitched up. Good recovery.

(*b*) J. P., æt. 21, miner. Fall of coal on head. Wound and depression in skull. Sent into hospital next day, after being dressed by doctor. Wound gave growth of staphylococci. Operation. Depressed bone elevated. Wound cleansed with 1 : 20 carbolic. Good recovery.

11. *Dr Charles M'Neil* showed—

Types of CHRONIC DYSPEPSIA IN CHILDHOOD amenable to treatment by emulsion of liquid paraffin: (*a*) Eleanor R., æt. $1\frac{4}{12}$ years,

chronic diarrhœa, with marked wasting; (*b*) James G., æt. 4 years, recurrent bilious attacks; (*c*) Thomas G., æt. 10½ years, nocturnal enureses, and malnutrition.

12. *Mr Charles W. Cathcart* showed—

RECOVERY OF WALKING POWER, after complete paraplegia following fracture of lumbar spine. No operation.

13. *Dr Boyd* showed—

(*a*) A case of BRONCHIECTASIS to illustrate oxygen replacement and artificial pneumothorax.

(*b*) A case of DIABETES WITH ACIDOSIS to illustrate benefit which may follow "oatmeal cure."

14. *Mr Lewis Beesly* showed—

Girl, 2 years old. TUBERCULOUS DACTYLITIS at 9 12. Abscess formation. Removal of sequestra with complete local recovery. 5 months later, painless enlargement around the left ulna. Tuberculous osteomyelitis in the proximal half of the diaphysis. Subperiosteal resection on July 14, 1913. Primary union, and no further sign of tuberculosis.

Radiograms by Dr Edmund Price.

15. *Mr Scot Skirving* showed—

Man, aged 31, after very extensive operation 4 months ago for SQUAMOUS EPITHELIOMA OF LEFT UPPER JAW. Owing to implication of the skin of the cheek, no flap could be made use of. Accordingly, parts involved (including left eye and part of nose) removed *en masse*, with the result that huge open cavity left. This cavity now much smaller, but still very large. A plastic operation to restore the mouth has been done. A further plastic operation to close the cavity in the side of the face to be carried out shortly, by a flap from scalp or left upper arm.

Much help obtained at operation from intratracheal method of giving the anæsthetic, and from preliminary ligature of external carotid.

Meeting IX.—June 3, 1914

Dr John Playfair, *President, in the Chair*

I. Photographic Demonstration

By Dr Chalmers Watson

Series of X-ray Photographs (illuminated) from cases of indigestion, associated with "rheumatism" or chronic ill-health, in which the X-ray examination revealed well-marked ileal stasis due to adhesions in the ileo-cæcal region, the X-ray findings being confirmed at the operation. (Radiograms by Dr Hope Fowler.)

II. Original Communications

1. JACKSON'S MEMBRANE AND THE GENITO-MESENTERIC FOLD OF PERITONEUM

By Douglas G. Reid, M.B., Cambridge

[The figures indicate the photographs and other illustrations shown to the Society of the specimens which are here described.]

Jackson's Membrane

Specimens 1 to 12 all present arrangements which bear upon Jackson's membrane.

1. In this fœtus there is an early stage of cæcal torsion, for the concavity of the cæcum looks towards the right. The "bloodless" fold of Treves, and the terminal part of the ileum, adhere to the parietal peritoneum and would resist the completion of torsion.

Such adhesions, and the genito-mesenteric fold, explain "the unexpected positions in which, at times, the appendix is found on opening the abdomen" (H. Macnaughton-Jones, "The Bearing of Sex on Operations in Appendicitis," *Med. Press and Circ.*, 1913, xcv. p. 196. For photographs showing the various stages of cæcal torsion, etc., see my paper, "Chiefly concerning the Genito-mesenteric Fold of Peritoneum," *Proc. Roy. Soc. Med.*, Feb. 1914).

2. *Adult.*—It is noteworthy in association with the adhesion, present in this specimen, of the fold of Treves to the patietal peritoneum, that cæcal torsion has not been completed.

3. *Adult.*—(See J. R. Eastman, "Fœtal Peritoneal Folds," *Journ. Amer. Med. Assoc.*, 1913, lxi. p. 635.) It is possible therefore that the cæcum may wrap itself up in Treves's fold (see also

specimens 31, 32, 33). It here forms a pocket from which "I have shelled the caput coli and appendix in operations for so-called appendicitis several times."

This gives us one explanation of the origin of a Jackson's membrane, and another interpretation of C. H. Mayo's view regarding its congenital origin. In this specimen the genito-mesenteric fold passed from the terminal part of the ileum to the ovary and Fallopian tube.

4. In this full-time fœtus the ascending colon has been acutely bent upon itself (see Fig. 1), undoubtedly through the pressure which has been exerted upon it by the pelvic colon.

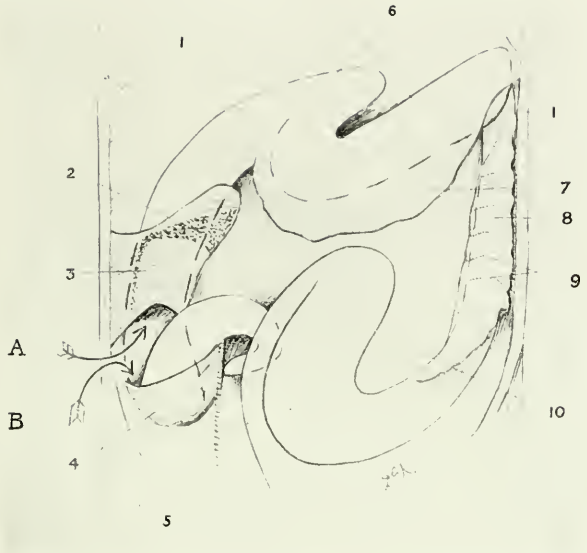
As is frequently the case in the fœtus, this forms a relatively enormous intra-abdominal loop. It lay in front of the inverted cæcum. The pressure which it exerted has also caused the lower part of the genito-mesenteric fold to be displaced towards the right off the line of the spermatic vessels, which primitively always lie along its posterior border. This pressure has caused the fold to form a pocket (see Fig.) in which the flexure is lodged. Moreover, it has caused the fold to adhere to the bowel, and the acute flexure is thus rendered permanent.

When the colon was shelled out of this pocket the fold was seen to be j-shaped. It first descended upon the spermatic vessels and then arched outwards over the right iliac fossa.

In relation to Jackson's membrane Charles Mayo suggested (see *Surg., Gyn., and Obstet.*, March 1911) that the cæcum might "burrow its way" down behind the parietal peritoneum. This would explain "that form of Jackson's membrane which encloses the cæcum and ascending colon as in a bag" (Eastman), and we have shown that the cæcum may actually come to lie in a pocket formed by Treves's fold, and that the genito-mesenteric fold may also wrap itself over the bowel and form a definite pocket.

5. *Fœtus.*—The paricto-colic fold of Jonnesco crosses in front of the ascending colon (see Fig. 1). It is a large peritoneal fold (appendix epiploica), bounds a pre-colic fossa, and is capable of resisting considerable traction.

6. *Adult.*—A strong and extensive Jackson's membrane is present. It crosses in front of the ascending colon to reach the lateral parietal peritoneum. The colon is acutely kinked at the hepatic flexure; but the ascending colon presents none of the angulations to which Jackson has drawn attention (see "Membranous Pericolitis," *Surg., Gyn., and Obstet.*, Sept. 1909). It is



1. Side wall of abdomen.
2. Area of adhesion.
3. Fold crossing ascending colon.
4. Genito-mesenteric fold forming pocket (full-time fœtus).
5. Spermatic vessels.
6. Transverse colon.
7. Great omentum.
8. Membrane coating descending colon (fœtus 17 cm. long).
9. Blood-vessels in membrane.
10. Pelvic colon ("compressed omega" form).

FIG. 1.—This figure was constructed from three fœtuses (17 and 18 cm. long [vertex to coccyx] and full-time) dissected by me at Cambridge. The ascending colon is bent upon itself, and the flexure lies in a pocket formed by the genito-mesenteric fold displaced to the right by the pelvic colon (pushed to the left off the front of the cæcum). Crossing the ascending colon is the parieto-colic fold. It is formed by a strong appendix epiploica and bounds a pre-colic fossa whose orifice is indicated by arrow A. Lying in front of, and adhering to, the descending colon is an extensive membrane formed by an appendix epiploica. It has a free left border which lies in contact with, but does not adhere to the lateral abdominal wall. Arrow B indicates a slight fossa between the genito-mesenteric fold and the lateral part of the colon.

PLATE I.—TO ILLUSTRATE ARTICLE BY MR DOUGLAS G. REID.

constricted to a remarkable degree, and both the longitudinal and circular layers of its muscular coat are much thicker than normal. The cæcum is neither "prolapsed," hypertrophied, nor dilated; and the terminal part of the ileum is free from adhesions.

Some say that "ptosis" of the cæcum is common in association with Jackson's membrane; but it was not present in any of the cases here referred to in which I found a well-marked membrane. The membrane contains fat, and, as in the fœtus just mentioned, blood-vessels which are visible macroscopically and lie parallel to one another and at right angles to the long axis of the bowel, across which the arteries extend to reach the lateral parietal peritoneum (see also *Journ. of Anat.*, July 1914, xlviii.).

There can be little doubt as to how this membrane arose.

7. *Adult.*—In this specimen I found a peritoneal fold lying in front of the ascending colon in practically its entire length. It has no connection with the great omentum, and has all the characters of an appendix epiploica which it undoubtedly is.

It contains fat and blood-vessels, visible macroscopically, and having the same disposition as in the case just recorded. Had it fused down to the bowel (and in relation to the upper and inner part of the ascending colon this had actually occurred to a slight extent) an exactly similar membrane would have been produced. The smaller appendices epiploicæ behind the fold would have given rise to "spots and tags of fat" (Hall).

8. In adults I have seen still larger folds of peritoneum (undoubtedly appendices epiploicæ) in relation to the ascending colon.

It is very common for appendices epiploicæ to adhere to the colon and form bands which usually lie at right angles to the long axis of the bowel. I have frequently seen them crossing the colon to adhere to the lateral parietal peritoneum; but only in one case was there a distinct constriction of the bowel associated with a strong but relatively narrow band formed in this way.

In three out of twelve adults whose abdomens were dissected at Cambridge this term (May 1914) I found extensive folds of peritoneum lying in front of the descending and iliac colons. In all three specimens they arose from the peritoneum exactly along the inner border of the colon. In one the fold extended from the splenic flexure to the pelvic brim and was considerably broader than the bowel. It had no connection with the great omentum. In another the fold, 25 cm. long, overlapped another fold almost half as long. It is extremely common to find these enormous folds

in this situation. The largest I have seen was 38 cm. long, and the descending and iliac colons were almost completely concealed behind it. All contained fat and blood-vessels, visible macroscopically and coursing at right angles to the bowel. They were undoubtedly appendices epiploicæ.

Had they adhered to the descending colon they would have prevented, or at all events hampered, the expansion of this normally relatively narrow part of the large intestine.

9. Fœtus, seven months old (see J. R. Eastman, "The Fœtal Peritoneal Folds of Jonnesco, Treves, and Reid, and their Probable Relationship to Jackson's Membrane and Lane's Kink," *Surg., Gyn., and Obstet.*, April 1913, xvi.). In this specimen there was "an extensive parieto-colic peritoneal fold constricting the lumen of the ascending colon" in front of which it crossed.

10. In this adult I found another strong and extensive membrane lying in front of and associated with marked constriction of the ascending colon. This at one point was only 22 mm. broad. The membrane adheres to the bowel, but prolonging its right border is a large free fold of peritoneum. This is an appendix epiploica.

A remnant of the genito-mesenteric fold is present, and fixes the terminal part of the ileum. Between the point so fixed and the ileo-cæcal junction the ileum seems to have become too long to lie in a straight line.

At all events it forms a gentle curve convex upwards. It is not acutely kinked, however.

11. In the same adult there is an extensive membrane investing the descending and iliac colons. It adheres to the bowel, but its left border is prolonged by free folds. *I have seen an exactly similar membrane in a fœtus 17 cm. long* (see Fig. 1). In this fœtus the free left margin of the membrane, the large arteries and veins (having the typical direction), and the bundles of non-medullated nerve fibres lying definitely in the membrane were especially noteworthy. There can be no doubt as to how these membranes arose. They are formed by one or several appendices epiploicæ.

Although these membranes and folds and their main blood-vessels lie at right angles to the long axis of the bowel, an appendix epiploica which crossed in front of the ascending colon would tend to become oblique did it adhere to the parietal peritoneum before the cæcum had completed its descent. The vessels would also become oblique. In this adult a strong appendix epiploica passes from one part to another of the transverse colon. It forms with the

bowel a foramen through which I could readily thrust a loop of small intestine.

12. *Adult* (see Eastman in paper just cited).—I have shown that in the fœtus the laminæ of the great omentum may fuse together to a remarkable extent, and that the great omentum may gain an attachment to the ascending colon and cæcum and pass from these to the parietal peritoneum (see *Journ. Anat. and Physiol.*, 1910-11, xlv. p. 73). In this specimen there was a membrane "formed by fusion of the normal peritoneum of the colon with the anterior and posterior laminæ of the great omentum." I have seen similar well-marked membranes in adults.

In one the right part of the transverse colon was closely bound to the upper part of the ascending colon simply by this omentum, which crossed between them. Jackson ("Membranous Pericolitis," *Surg., Gyn., and Obstet.*, Sept. 1909) notes that the transverse colon may be "drawn down" to the ascending colon by the "membrane."

It will be seen that specimens 1 to 12 afford us explanations of the origin of Jackson's membrane apart from pathological causes.

Professor Joseph Rilus Eastman first drew attention to my pre-colic folds in relation to Jackson's "membranous pericolitis," about which I was ignorant at the time I published my papers.

The Genito-mesenteric Fold of Peritoneum

13. *Full-time Fœtus.*—In this specimen the genito-mesenteric fold, which I have described in the fœtus (see *Journ. of Anat. and Physiol.*, 1908-9, 1910-11, 1911-12), and demonstrated in adults, passes from the mesentery downwards to the right ovary and Fallopian tube.

It is noteworthy that the ileo-appendicular fold of Jonnesco ("bloodless" fold of Treves) is completely separate from the genito-mesenteric fold.

Frequency.—The fold was present in eleven (six males and five females) out of twenty 12 to 22 cm. (vertex-coccygeal measurement) fœtuses (55 per cent.). It occurred in seven out of ten (70 per cent.) of full-time fœtuses recently examined, and must be fairly common in the child. As the fold is a track by which infection or inflammation may spread from the bowel (appendix, ileum, cæcum) to the ovary and Fallopian tube, or in the opposite direction, it is noteworthy that not only is appendicitis common

in children but that numerous cases have been recorded in them of salpingitis, "sometimes accompanied with ovaritis" (see *e.g.* Pfaundler and Schlossmann, *The Diseases of Children*, iii., and H. A. Kelly, *Operative Gynecology*, 1906, ii. p. 566). The genito-mesenteric fold was present in six out of eighteen adults (33 per cent.).

Genesis of the Genito-mesenteric Fold (?).—Eastman has suggested "that in the descent of the right ovary or testis the dorsal peritoneum is caught and dragged or drawn out into this form."

It is difficult to say exactly how the fold arises.

One must remember its relation to the ileac branch of the ileo-colic artery (see legend, Fig. 2), and that it appears after the fourth month of intra-uterine life. Apart from the blood-vessels, I found no muscular fibres in a portion of a fold removed from a full-time fœtus. (It may, however, be desirable to examine folds in fœtuses of various ages.)

14. *Full-time Fœtus.*—The genito-mesenteric fold passes from the terminal part of the ileum downwards towards the right ovary. The lower part of the fold is displaced slightly towards the right and just fails to become continuous with the plica vascularis. This interruption has undoubtedly been produced by the pressure which the large meconium distended pelvic colon has exerted upon the fold. It has caused the lower part of the fold, with which it lay in contact, to adhere to the posterior parietal peritoneum. We have now seen that this part of the ileum may be connected to the abdominal wall by (*a*) the genito-mesenteric fold, or (*b*) the bloodless fold of Treves.

15. *Fœtus.*—I think that some of my friends may have confused (*a*) with (*b*). (*b*) may adhere to (*a*) as in this fœtus.

16. In this adult (*b*) also adheres to (*a*). (*a*) passes between the terminal part of the ileum and the right Fallopian tube and ovary. Through this adhesion (*b*) connects the ileum and appendix to the ovary and tube.

The connection of the ileum with the genito-mesenteric fold is a secondary one.

In 14, as in other specimens, the fold can be traced over the surface of the bowel, to which it adheres, to the under surface of the mesentery.

17. In this fœtus, as in some others, the genito-mesenteric fold forms an extremely short connecting link between the ileum and the right ovary and Fallopian tube.

Photograph by] [*Mr W. J. Calcott.*

FIG. 2.—Full-time fœtus, o. The cæcum and ascending colon were "free" and have been pulled forward. The genito-mesenteric fold is viewed from the right side. It extends from the mesentery and duodenum to the internal abdominal ring. Its mesenteric border is related to the ileac branch of the ileo-colic artery; and the spermatic vessels and nerves (spermatic plexus, external spermatic nerve) and the external iliac artery lie along its parietal (posterior) border. Vessels have been carried on to the right surface of the fold by the great omentum. The appendicular artery is often bound to this surface. Occasionally it lies along the anterior border of the fold. The middle strip of white paper lies between the appendix and meso-appendix, and the terminal part of the ileum and "bloodless" fold of Treves, none of which adhere to the main fold. There was a large retro-pancreatico-duodenal fossa. (Fœtus dissected by me at Cambridge.)

PLATE II.—TO ILLUSTRATE ARTICLE BY MR DOUGLAS G. REID.

18. *Full-time Fœtus.*—In this specimen the dark objects in the genito-mesenteric fold are lymph-nodes. I have demonstrated their presence in the fold in other fœtuses. In some cases the great omentum may carry blood and lymph-vessels on to its right surface (see Fig. 2).

These points we consider important in relation to the spread of infection.

19. *Full-time Fœtus.*—The common and secondary connection between the meso-appendix and genito-mesenteric fold is of importance.

20. *Fœtus.*—Through it, or through an adhesion of the appendix directly to the fold, the appendix may be connected to the right ovary and Fallopian tube as in this specimen ; and it is possible that infection or inflammation may spread by this route, from the appendix to the right (or left, see Macnaughton-Jones in paper already cited) ovary and Fallopian tube, or in the opposite direction.

21. *Fœtus.*—The vermiform appendix, indeed, may be most closely bound to the ovary and Fallopian tube by the fold. In such a case some gynæcologists might think themselves justified in removing the normal appendix as a prophylactic measure (see H. Macnaughton-Jones, "The Relation of the Appendix to the Causation and Surgical Treatment of Affections of the Adnexa," *Proc. Roy. Soc. Med.*, 1911, iv. p. 346).

The appendix may be firmly held in a retro-colic position by the genito-mesenteric fold, as I have noted in the adult and fœtuses.

22-3. In this specimen (a full-time fœtus) the ileum adheres to the right surface of the genito-mesenteric fold, *as does the appendix.* They both form flexures which are thus rendered permanent. Their lymph-vessels are thrown into immediate connection. This, however, occurs frequently, as the ileum often fuses down over the meso-appendix to the parietal peritoneum. The lower part of the genito-mesenteric fold has been displaced to the right and forms a shelf, concave upwards, upon which the appendix rests. The shelf (sometimes there is a definite pocket) also adheres to the appendix. These adhesions were undoubtedly determined by the pelvic colon which forms a large loop lying in contact with the fold.

I have noted that the appendix and its mesentery may be acutely kinked through being bent over the free, sharp edge of the genito-mesenteric fold.

Inflammation, perhaps strangulation, might follow through interference with its blood-supply. The ileum might also become strangulated in a somewhat similar manner (see my paper in the *Journ. of Anat. and Physiol.*, 1913, xlvii. p. 470).

24. *Fœtus.*—The genito-mesenteric fold is often (usually in females) continuous with the suspensory ligament, or plica vascularis, of the genital gland.

25. *Adult Male.*—We have an explanation, therefore, of how the fold is here attached to the peritoneum at the internal abdominal ring.

26. In this adult the genito-mesenteric fold was seen "during an operation for a pelvic lesion" (Eastman).

It was not associated with a kink of the terminal part of the ileum and "produced no symptoms."

Eastman has also seen the genito-mesenteric fold "associated with inflammation and contracture," and producing a kink of the ileum (*Personal Communication*, Feb. 1913).

27. *Adult.*—The cæcum, ileum, and meso-appendix adhere to the genito-mesenteric fold. This bounds a narrow retro-colic fossa, containing the appendix, which was best reached by pulling the ileum forwards and dividing the fold.

In cutting the fold one must not forget its relations to the appendix, meso-appendix, vessels, and nerves (see legend, Fig. 2).

We have now seen that the appendix, meso-appendix, ileum, and its mesentery, cæcum, and (6) fold of Treves may all become *secondarily* connected to the main (genito-mesenteric) fold.

28. In this fœtus the ileo-colic artery is seen lying in a fold of peritoneum which I have called *the superior or left-root fold*, since it lies at the root of the mesentery.

It may bound a definite fossa (*left or superior root-fossa*) large enough to contain a piece of small intestine and, indeed, a part of the pelvic colon, which often ascends to it (and to the duodenum) in the fœtus.

If the summit of the pelvic colon remain in this position, the mesentery may fuse down over the pelvic colon. I have seen this in several adults.

It is also noteworthy that if the "terminal intestine" (the part of the large intestine caudad to the splenic flexure—Jonnesco) rotate to the right, instead of to the left, the mesentery may fuse down over the descending, as well as the pelvic colon.

In colostomy these and other adhesions, such as I have seen, might cause considerable trouble.

Adhesions determined by the Genito-mesenteric Fold and Pelvic Colon

29-30-31. *Fœtuses.*—Through anchoring, and apparently exerting traction on the mesentery, the genito-mesenteric fold may cause this to adhere to the parietal peritoneum. It can thus lower the root of the mesentery even before the descent of the cæcum, and may determine most extensive adhesions in the ileo-cæcal region (I have noted this in a large number of fœtuses) and also displacements of the bowel, *e.g.* a downward displacement of the duodenum, right-sided congenital inguinal hernia (see my paper, *Proc. Roy. Soc. Med.*, Feb. 1914). The terminal part of the ileum is rendered relatively immovable through the connection of its mesentery with the fold, and its connection with the large intestine.

It is confined between the fold and the relatively unyielding abdominal wall; and the fold may be pressed against the ileum, cæcum, appendix, and ascending colon by the large pelvic colon. The ileum may readily become adherent to adjacent peritoneum, as may its mesentery.

Adhesions (perhaps better termed "cohesions"), such as I have described in the fœtus, may form barriers, ready-made, resisting the spread of inflammation. They explain retro-colic abscess (Macnaughton-Jones), etc.

32. *Adults.*—Adhesions binding the terminal part of the ileum directly to the parietal peritoneum of the right iliac fossa occur very frequently in the fœtus as well as in the adult. They may render truly permanent flexures of this part of the bowel.

33. In the fœtus, movements of the pelvic colon and other parts of the large intestine may occur which produce acute flexures, and these may then be rendered permanent by adhesions such as I have described in my paper (*Proc. Roy. Soc. Med.*, Feb. 1914).

34. *Full-time Fœtus.*—The sub-splenic loop is sometimes present in the adult as well as in the fœtus. It is an antero-posterior loop upon the upper part of the descending colon, and forms an acute and permanent flexure, for its limbs are firmly bound together, and in some cases they also adhere to the lateral parietal peritoneum.

The pelvic colon must also be kept in mind in accounting for adhesions. *E.g.* sometimes in adults one finds the appendix and meso-appendix bound directly to the under (left) lamina of the mesentery. I have found this, and adhesions of the appendix

and cæcum to the parietal peritoneum, produced in fœtuses by the pressure exerted upon these parts by the pelvic colon. They may adhere to the peritoneum over an area which in extent exactly equals the surface upon which the pressure was exerted.

35-6. *Fœtus.* - The pelvic colon may also press upon and cause a displacement to the right and obliteration of part of the genito-mesenteric fold (track by which infection or inflammation may spread). I have already indicated this and the fact that this pressure may cause the fold to adhere to the bowel. I have demonstrated (see *Journ. of Anat.*, 1911, xlv. p. 406) that the pelvic colon may even, by pressing upon its own mesentery, cause this to adhere to the parietal peritoneum. One should also remember that the pelvic colon may adhere to the duodenum, with which in the fœtus it often lies in contact. It may also determine, or help to determine, other adhesions.

Quite frequently in adults the second part of the duodenum is closely bound to the ascending colon.

Stages of Cæcal Torsion

37-8-9. *Fœtuses.* (*a*) See also specimen 1. The appendix is in front, then (*b*) on the outer side, and then (*c*) behind the ascending colon. (*d*) Sometimes it lies completely below the cæcum. In (*c*) and (*d*) the appendix may be fixed in these positions by a genito-mesenteric fold, and in (*d*) the appendix may later become retro-colic if the cæcum be able to complete its descent.

The Retro-pancreatico-duodenal Recess

40. *Fœtus.* The caudate lobe (process) of the liver lies in contact with the *posterior* surface of the head of the pancreas. Should this recess persist (I have found it well marked in children at birth and in one adult), inflammatory exudations, especially from the stomach or duodenum, might accumulate in it and inflammation spread to the head of the pancreas.

It is noteworthy that there is sometimes a distinct prolongation of the head of the pancreas that may extend upwards beyond the first part of the duodenum (see Charpy, *Poirier's Anatomy*, 1905, iv. p. 822). For other points regarding the duodenum see my "Studies of the Intestine and Peritoneum," Part vi., *Journ. of Anat. and Physiol.*, xlvii. I must here refer to Professor C. M. Jackson's paper "On the Topography of the Pancreas in the Human Fœtus," *Anatomischer Anzeiger*, Band xxvii., 1905. I

regret that when I wrote on the duodenum this interesting work had escaped my notice. In an adult I have noted a marked dilatation of the second part of the duodenum in association with the presence of a strong band of mucous membrane, which extended from the valvula connivens (plica circularis) which lies just above, and forms a hood for the bile papilla. It was attached to the right wall of the duodenum, and sub-divided the lumen of the bowel into two nearly equal parts. There were no evidences of inflammation, and the band most probably resulted from the partial persistence of the embryological atresia described by Tandler.

Other Fœtal Peritoneal Adhesions

41-4. *Fœtuses and Adults.*—The following are amongst some of the other adhesions found in the fœtus :—

(1) The first part of the duodenum and the transverse colon may adhere together and both become bound directly to the gall-bladder.

In some cases this may later give rise to a definite cysto-colic fold, through traction upon the connective tissue elements where they have become continuous. This cysto-colic ligament was present in 30 per cent. of twenty fœtuses. It is not more frequent in the adult. (It occurs in 30 per cent. Addison.) It explains how biliary calculi may be éliminés avec facilité par le côlon (see Bricon, *Le Progrès Médical*, vii. p. 27).

(2) In fœtuses 25 and 37 mm. long the postero-inferior surface of the stomach is "free." I found that after the fourth month adhesions form between the stomach (even occasionally the small omentum) and the transverse meso-colon. *These adhesions occur normally.* In the production of adhesions (cohesions) apart from inflammation there must be relative fixation as well as pressing together of peritoneal sheets ; and it appears that adhesions tend to form most readily whilst the parts are actively growing. In the fœtus and young child they are scarcely, if at all, less extensive than in the adult.

(3) The laminæ of the great omentum may become welded together to a remarkable extent.

In accounting for these adhesions the firm resistance of the large fœtal liver must be remembered (see also my papers already cited, and p. 267, *Journ. of Anat. and Physiol.*, xlvii.).

45. These adhesions (besides being, we think, of interest to the anatomist and surgeon for various reasons) help to form com-

partments or recesses (which are sometimes "definite ready-made *abscess cavities*"). For a figure showing these, see *Journ. of Anat. and Physiol.*, xliii. p. 506.

Discussion

The President felt that the Society was very deeply indebted to Mr Reid for his valuable paper and beautiful illustrations. Many of the statements made and ideas put forward were new, and perhaps required a specialist to discuss them and fully appreciate their value. But even the grasp obtained from the demonstration showed that many practical points had been brought out which threw light upon many hitherto obscure points. The paper would repay prolonged study.

Mr Cathcart regretted that his acquaintance with the anatomy of these folds did not enable him to appreciate the anatomical points brought out, but as a practical surgeon he was glad that many of the folds described are recognised as anatomical. In opening the abdomen he had frequently found these folds—Jackson's membrane and others—and thought that many of them did not seem to be pathological. It had always struck him that adhesions cannot be the whole explanation of the pathological conditions included in the term intestinal stasis; he thought it must be a question of peristalsis. One sometimes saw cases. Dr Wilkie had shown a great number of specimens where adhesions are present, where the intestine may be buried in adhesions, and yet no obstruction followed. He had never yet been convinced that adhesions pulling the bowel a little out of its course would necessarily lead to obstruction unless there was a loss of peristaltic power. The fact that Mr Reid's work was done so thoroughly would help to bring the point to a definite conclusion. They were very much indebted to him.

Mr D. P. D. Wilkie said the paper was specially valuable coming from one who looked at the subject from an entirely scientific point of view, and who had perhaps not been brought into discussions as to the exact pathological process which underlies adhesions in the abdominal cavity. The folds shown are very like some of those taken to be pathological. If he understood Mr Reid rightly, they were present in practically every fœtus. Were

they to understand that this genito-mesenteric fold is invariably present? Post-mortem the fold described is present in from 20 to 30 per cent. In the other cases one would like to know whether it had disappeared or fused or become unrecognisable? He had come to the conclusion that there were three distinct types of membranous folds met with in the region of the ileum and proximal colon: (1) the genito-mesenteric and the parieto-colic folds —undoubtedly developmental; (2) inflammatory adhesive folds resulting respectively from appendicitis and ascendens colitis; (3) the folds representing the crystallisation of lines of strain as described by Lane.

Further, he would like to ask Mr Reid whether in his observations on adult bodies he had drawn any distinction between such types, or did he regard them all as merely modified congenital folds.

Mr Scot Skirving thought the Society would like, in the first place, to know clearly whether Mr Reid considered the genito-mesenteric fold entirely congenital in origin. In the second place, it was stated in the précis that "the fold occurred in 55 per cent. of fœtuses, 70 per cent. of children at birth, 33 per cent. of adults." If it was entirely a congenital condition, why did it only occur in 33 per cent of adults?

Mr Beesly was quite in agreement with regard to the anatomical development of these folds. In regard to Jackson's veil he pointed out that this pericolic membrane was very rarely seen in the dissecting-room, whereas it was present in the majority of abdomens examined on the operating-table, both in children and adults. He suggested that it was a normal condition, and that its absence could be accounted for in the cadaver by the changes following death, especially by the hardening of the extra-peritoneal fatty layer. This hardening prevented the peritoneum slipping about easily within the abdomen, and comparison between the difficulty in the cadaver and the ease in the living subject with which the bowel could be lifted out of the abdomen was manifest and familiar to all.

Mr Douglas Reid in reply said the genito-mesenteric fold was not always present. Mr Scot Skirving referred to the percentages given. The absence of the fold was due to the pressure which had been exerted on it by the pelvic colon. The fold may be

obliterated. He had shown slides where pressure of the pelvic colon on the fold caused it to adhere. He had shown that other adhesions were formed in this way. Again, the mesentery might fuse over the upper part of the gonto-mesenteric fold and obliteration of the whole fold occur. That explained why the fold was not so common in the adult. It also explained how the root of the mesentery might be lowered. Whenever a part of the peritoneum is rendered immovable, adhesions tend to form. In the fœtus he had drawn special attention to the extension of the great omentum downwards upon the ascending colon and cæcum. He indicated how an appendix epiploica investing a still short ascending colon might become oblique consequent upon its adhesion to the abdominal wall and then the descent of the cæcum.

2. NOTE ON THE INTRATRACHEAL INSUFFLATION OF ETHER

By H. Torrance Thomson, M.D., Anæsthetist Leith Hospital and J. W. Struthers, F.R.C.S.Ed., Assistant-Surgeon Royal Infirmary, Edinburgh

Intratracheal insufflation consists in driving a current of air, under pressure, through a tube, introduced by way of the mouth and larynx, deeply into the trachea. The current of air returns between the tube and the wall of the trachea, and escapes through the mouth and nose. The current is continuous.

The work of Meltzer and Auer has demonstrated that this insufflation of air into the trachea under adequate pressure ventilates the pulmonary alveoli and enables the normal diffusion of gases to be carried out for many hours, independently of all respiratory movements. If the air in its passage under pressure is made to pass through a chamber containing ether, we are enabled to introduce into the pulmonary alveoli ether vapour of varying strength.

The points to which attention may be directed are:—

1. In ordinary respiration air has to be brought from outside through the nose, pharynx, larynx, trachea, and bronchi—from this point respiration consists in an interchange of gases between the tidal and alveolar air by a process of diffusion.

In intratracheal insufflation, air is brought down to the bifurcation of the trachea, with a certain force which favours diffusion of gases, the long intervening air-shaft which has been termed the

dead space being thus cut out. The practical importance of this is that there can be no interference with the free passage of air between the entrance to the mouth or nose and the trachea, an interference which is so frequently responsible for respiratory difficulties during anæsthesia.

In addition, an excellent means for performing artificial respiration is always available. As an instance of its utility in this respect, reference may be made to a case recorded by Elsberg, in which in a patient who had taken morphia with suicidal intent, artificial respiration was kept up for 12 hours, without any respiratory movements taking place, recovery ultimately ensuing. In our own experience at Leith Hospital, the apparatus was used successfully in a patient who had attempted suicide by drowning, and in whom respiratory movements had ceased. Artificial respiration was kept up for several hours, after which ordinary respiratory movements were re-established.

2. In intratracheal insufflation there is, during the entire period, a continuous current of air blowing forcefully from the trachea through the larynx, pharynx, nose, and mouth. It has been demonstrated in the laboratory and in actual practice that, during insufflation, it is impossible for blood, mucus, or vomited matter to enter the larynx, obviously a point of great practical importance in many operations.

3. By the constant maintenance of sufficient positive pressure, collapse of the lungs may be prevented or regulated at will, when the thorax is opened. In virtue of this and because the method does not depend on the efficiency of the patient's respiratory movements, intratracheal insufflation of ether appears likely to displace the elaborate negative and positive pressure cabinets and masks which have been devised for intrathoracic operations.

The claims made on behalf of the method are based on the records of a large number of laboratory experiments carried out by Meltzer, Auer, and others, and on the observations of anæsthetists and surgeons in actual practice.

Our experience is derived from the use of the method in about 100 cases. The cases were not specially selected, our object being to gain experience as quickly as possible and to acquire dexterity in the technique—so that special cases might have a fair trial. The series, however, included many cases which afforded opportunity to put the method to the test with regard to the special advantages claimed for it.

Among the cases coming under this category, the following may be specially mentioned :—

Laparotomies for Gall Stones—intraperitoneal rupture of bladder, etc.
Œsophagostomy.
Œsophagoscopy.
Operations for Mastoid Disease.
,, Tubercular Glands in Neck.
,, Nasopharyngeal Polypus.
,, Strangulated Hernia.
Prostatectomy.
Nephrectomy.
Lainmectomy.
Excision of Upper Jaw.
Excision of Tongue.
Plastic Operation Cheek.
Plastic Operation Mouth.
Hysterectomies in Irendelentomy Position.

To commence with, we used an apparatus made after the model described by Meltzer. It is inexpensive and efficient.

For the greater number of our operations we have used the apparatus devised by Mr Kelly of Liverpool. This consists of two parts, the first part being for the production of the air current —the second for the etherisation of the air. The electric motor actuates a rotatory blower which produces a current of air. The blower runs at the rate of from 50 to 1000 revolutions per minute. The ether apparatus consists of an ether chamber, 12 cm. in diameter —metal lid, with entry and exit for air current, and third opening for introduction of ether. The entry tube dips down into the air space above ether for about 4 cm. Entry and exit tubes are interrupted by taps, geared by cogwheels to a third tap connecting both entry and exit. This permits of variation in ether strength of air current from zero to full saturation. In addition there are (1) warming chamber, (2) safety-valve, (3) mercury manometer, (4) three taps, (*a*) pressure regulator, (*b*) alternative tap for oxygen or NO_2 gas, (*c*) tap for reducing pressure in trachea to zero.

The preliminary administration of morphia, gr. $\frac{1}{4}$, and atropine, gr. $\frac{1}{100}$, has been the rule. In alcoholic subjects, it would probably be an advantage to add scopolamine, gr. $\frac{1}{100}$.

Induction is carried out in the ordinary way.

For the introduction of the catheter we have used Hill's Endoscope. The passage of the catheter is at first a matter of difficulty —but with practice it becomes comparatively easy. The important points to attend to are (1) thorough relaxation before attempting intubation—the lower jaw should be so slack that a gag is not required ; (2) the head should hang over the end of the table or be placed in the occipito shoulder position ; (3) the epiglottis must be identified—and the speculum passed sufficiently far below its tip that it will not slip—too deep insertion must be avoided ; (4) keep speculum in left hand, and with a tilting movement of the hand lift the hyoid bone. The glottis then comes into view, and there should be no difficulty in inserting the catheter, which should be made rigid by means of a probe. We have used the silk-web, white-enamelled cylindrical catheters. They stand boiling fairly well if care be taken not to leave them too long in the boiling water, and if each catheter is wrapped in a piece of gauze so that it is not in contact with another catheter. The instrument is pushed nearly to the bifurcation of the trachea, a point in the adult about 26 cm. from the incisor teeth—the probe is withdrawn and connection made with the air current. The size of the catheter varies with the build of the patient. In all but exceptional cases, our experience leads us to believe that the maximum size should be 24 F., ranging downwards from this. Too large a catheter will interfere with the return current of air.

Occasionally the tube has been passed into the œsophagus ; this occurred several times in our earlier cases. With care and adequate relaxation such a mistake should not occur, but the possibility of it should be kept in mind. If the patient has been properly anæsthetised and the tube introduced without delay any glottic spasm which supervenes should rapidly pass off. The degree of concentration of the vapour should not be too high to commence with, or irritation will result, evidenced by spasm and coughing. The pressure should vary according to the case from 10 up to 30 mm. Hg. The safety-valve should be so arranged as to make any pressure above this impossible.

In the majority of cases, the course of anæsthesia has been perfectly smooth and uneventful—the colour remained a rosy pink, the pulse good, and the respirations quiet and regular. There was a noticeable absence of strain of all kind, the condition resembling natural sleep rather than narcosis. As a rule, the anæsthesia is light and easily controlled by turning the regulator either

towards full ether or air, and at any moment the ether can be turned off altogether, the apparatus being at once converted into one for performing artificial respiration.

It is undesirable that the respiratory movements should be abolished altogether, their presence indicates that the pressure of the entering air is not too great for the respiratory muscles, weakened by the anæsthetic, and also that the respiratory centre in the medulla is not being overdosed with ether. At the conclusion of the operation, before withdrawing the catheter, it is well to flush out the lungs with air so as to remove any ether vapour that is present. This is accomplished by turning the regulator to air for a few minutes. In a few cases, notably in big alcoholic subjects, difficulty has been experienced in securing a sufficiently deep anæsthesia with good relaxation. In two of our cases the attempt had to be abandoned, and anæsthesia maintained by other methods. The attempt in such cases to give a more concentrated vapour has in our experience led to coughing and spasm. Probably, as previously suggested, the addition of scopolamine to the preliminary dose of morphia and atropine would be advisable, and it is particularly important to produce a deep anæsthesia in such subjects before commencing insufflation.

There has not been any case of sickness during the insufflation. With such a limited experience to draw upon, it is difficult to speak with any assurance regarding the question of shock. The absence of strain and the perfect aeration should certainly help to lessen shock, and the general opinion appears strongly to favour the view that it does so. In our series, there was one case of bronchitis. In a few cases, there was some hoarseness and complaint of sore throat, more marked in the early cases and possibly due more to clumsy introduction than to irritation from the intratracheal tube. There has been no case of pneumonia.

After-sickness occurred in a certain proportion of the cases; it is generally held by those who have had experience of the method that the amount of after-sickness is less than after ordinary inhalation anæsthesia. In the meantime, we refrain from expressing an opinion on this point.

In estimating the value of this method, it is in the first place to be remembered that it is essentially a method of maintaining anæsthesia, it does not touch the problem of satisfactory induction. The general conclusions to which one came may be briefly summarised. Provided a satisfactory apparatus is used

and reasonable precautions are taken, the method is a very safe one. The precautions referred to are concerned with such points as the skilful introduction of a properly sized tube, the regulation of the pressure, and the maintenance of respiratory movement.

In prolonged operations the method provides a quiet, even anæsthesia with perfect aeration and marked absence of all strain. One is struck by the peaceful nature of the anæsthesia, the condition as previously remarked resembling a natural sleep rather than a narcosis. In abdominal operations, the quiet respiration and the relaxation of the muscles of the abdominal walls make the method a very satisfactory one, especially in operations in the upper part of the abdomen. It promises to relieve the respiratory embarrassment which is sometimes seen during the course of anæsthesia in stout patients who are placed in the Trendelenberg position.

In all operations about the mouth, such as those for excision of the upper jaw—antrum operations—or those undertaken for removal of nasopharyngeal growths, any danger of aspiration of blood mucus, etc., is removed, and the anæsthetist is well out of the surgeon's way. There is no interruption to the regular delivery of ether vapour, which is so liable to occur in such cases with ordinary methods. In operations for removal of glands in the neck or for mastoid disease, the surgeon has the field to himself, and is not hampered nor is his asepsis endangered by the proximity of the anæsthetist's mask. In such operations as laminectomy and nephrectomy, the postural difficulties with which the anæsthetist has to contend, and which also tend to interfere with free respiration, are eliminated. We have unfortunately not had the opportunity of testing the method in thoracic surgery. The opinion has been expressed that it will supersede the positive and negative pressure cabinets and greatly extend the possibilities of intrathoracic operations.

In conclusion, while anxious to avoid making extravagant claims, we suggest that, in intratracheal insufflation of ether, we have a very safe method of maintaining anæsthesia which in properly selected cases offers great advantages, and that the method deserves to take its place alongside of other methods at the disposal of the anæsthetist.

Discussion

The President said they had listened with very great pleasure and profit to Dr Thomson's paper. It was extremely ingenious,

and seemed to be a valuable method of administering ether and also for artificial respiration. It was rather an elaborate apparatus to carry about; it would be most valuable in an institution such as the Infirmary. The production of the apparatus must have entailed a great amount of thought and work.

Mr Hodsdon had had a limited experience of the method. It had been used in about 50 cases in his wards, and he had every reason to be satisfied. He had not as yet used it in intrathoracic surgery. There had been only one case of slight bronchitis, and that occurred after an operation for goitre—a class of case in which the method was extremely successful. The ward sister thought that after-sickness was less than when other methods had been employed. The usual procedure adopted was to give the patient scopolamine and morphia, then to administer a general anæsthetic, and finally to introduce the intratracheal tube. It was extremely important that the patient should be completely anæsthetised before the tube is introduced.

Mr Caird expressed his appreciation of the paper and of the method described. He believed the method would prove specially valuable in the case of cerebral cures with threatened failure of respiration, as also in certain cases where it was necessary to enter the thoracic cavity. In regard to its general application he was doubtful, since he felt that the methods for attaining anæsthesia would not be too simple.

Intratracheal tubes demanded special dexterity, were not free from danger, and might fail. Such methods had formerly been largely adopted in operative procedures involving the pharynx, but had been tacitly abandoned. More recent methods of anæsthesia had led to their revival, and by intratracheal tubes, by various negative and positive pressure devices for mechanical artificial respiration, the field of pulmonary surgery had been extended. He still felt, however, that the methods described should be restricted to such cases, for, apart from the difficulties of introducing a tube through the larynx, none of the special methods employed were entirely free from the risk of damaging the parenchymatous tissue of the lungs. He had observed that the majority of surgeons who had such forms of apparatus at their disposal dispensed with such aid as far as possible and reserved it for special cases.

Mr Scot Skirving thought it would be discourteous to his Leith colleagues if he did not, in a word or two, express his opinion of this method. In the first place, he had, so far, seen no evil result follow. Secondly, in certain cases he had received very obvious benefit from its use. Two most successful cases had already been referred to. One was an excision of the upper jaw in which a very wide resection had to be carried out. During the operation one worked in a puddle of blood with the utmost comfort. When the rubber tube was withdrawn there was not a stain of blood upon it, proving that no blood had gone down the trachea. Of course it might be asked, Would you not have got the same result if you had used the ordinary methods? The second case proved the contrary. It was that of an alcoholic dock labourer. He had an abdominal hernia so large that two fists could be put into the gap. There was the greatest difficulty in getting the recti together; personally he could not recollect any case in which there were more rigid recti. The patient was blue, he was coughing, and one even feared the operation might not be completed. As soon as the intratracheal method was substituted, the abdomen became relaxed and the patient's breathing became quiet.

Turning to the drawbacks to the method, Dr Thomson had referred to the risk of passing the tube down into the stomach, and had said that this mistake was *occasionally* made. He (Mr Scot Skirving) thought that the ordinary medical man would put it 5 times out of 6 into the stomach. Then the elaborateness of the apparatus was a disadvantage, and its size. One did not want to have to take a cab every day when called upon to give an anæsthetic! Mr Caird, he thought, had been too pessimistic in his remarks. It was a method which would in many cases be very useful in the hands of the anæsthetic specialist, but was not a method for the general practitioner. He did not think it would share the same fate as many previous methods, and it was one which surgeons would be extremely glad to have at their disposal.

Dr Stuart Ross said, in the first place, he would like to know whether Dr Thomson used this method in children. The youngest child he had used it on was 10; he confessed he was a little nervous for the first few minutes. He would like to hear Dr Thomson's experience on that point. With regard to introducing the catheter, he had a great deal of difficulty in his earlier cases and had adopted the plan of painting the pharynx with novococaine.

One could get the catheter in under a quite light anæsthesia, and he now had no serious difficulty. Another point is that sometimes one found a big, strong man can be overcome quite easily, while at another time there is the greatest struggle to keep even an average patient anæsthetised. This he attributed to differences in ether percentage, even when the indicator was at "full ether." Mr Alexander had kindly allowed him to work out the percentage of ether with the glass full and at different levels. If it is three quarters full, one gets 17 per cent.; if half full, 13 per cent.: and if one quarter full, 7 per cent., which latter is not sufficient to anæsthetise the patient. He was inclined to think that the ideal type of instrument would have to be made somewhat differently. He did not think that the method of blowing air over the surface of ether is the best possible way of making ether vapour. Again, when Dr Thomson had a case in which he finds the anæsthesia is not as deep as he would like and wishes to deepen it, did he regard increasing the tension of the atmosphere or increasing the ether percentage the more reliable? Obviously one wished to use as little ether as possible. He had asked the nurses in the Deaconess Hospital, whom he regarded as the best judges, as to the after-results; they were unanimous in their opinion that no other method of giving ether gave such good results.

Dr Burn Murdoch said he took it that the tube went into the larynx. How was the pressure given. He did not suppose that the trachea was blocked up each time. Was the pressure inside the lung?

Professor Bradley asked if the apparatus could be used for chloroform?

Dr Torrance Thomson in reply said that Professor Caird seemed to be under the impression that he proposed to use this method in all cases—whereas he had particularly stated that he thought it would take its place alongside of other methods in properly selected cases. The device to which Mr Caird referred of passing a catheter into the trachea and allowing the patient to inhale chloroform through it was of course not insufflation. Mr Caird was of opinion that the introduction of a catheter into the trachea could not but be harmful. His (Dr Thomson's) experience, though not large, did not agree with this, and the experience of

those who had worked in the laboratory and in actual practice had not borne out that suggestion. In Meltzer's laboratory experiments, that point was very carefully investigated, and the conclusion was arrived at that even after prolonged retention of the catheter in the trachea—say 12 hours—no irritation resulted. Dr Thomson's experience in children was limited to one case—and in that case Dr Logan Turner passed the catheter—a soft rubber one ; the anæsthesia was quite satisfactory.

As regards increasing the strength of the vapour—in addition to allowing the full saturation of the air by turning indicator to ether, the strength might be increased by immersing the ether chamber in warm water—Kelly's latest pattern provided for this.

Of course, the ideal was to have an instrument which would record the strength of the vapour, and this doubtless would be forthcoming. Increasing the pressure probably would diminish the strength of the vapour.

As regards Dr Burn Murdoch's question, one did not get an intrathoracic pressure equal to that recorded by the manometer.

Mr Struthers also replied.

Meeting X.—July 1, 1914

Dr John Playfair, *President, in the Chair*

I. Exhibition of Patient

Dr Norman Walker showed—

Case of lupus showing the result of treatment by tuberculin.

2. REPORT BY THE MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH ON ISOLATION AND OTHER MATTERS IN CONNECTION WITH THE MORE COMMON INFECTIOUS DISEASES

The following Report was drawn up by a Committee appointed by the Council of the Medico-Chirurgical Society of Edinburgh at the request of that Society. The Committee consisted of Dr Burn-Murdoch, Dr Melville Dunlop, Dr Maxwell Williamson (M.O.H.), Dr Meikle, Dr Claude Ker, and Mr J. M. Cotterill (*Convener*).

The Report was discussed and finally adopted by the Society at a meeting held on July 1st, 1914, with the additions (printed in italics for identification) occurring in Section II., paragraph B, and in Section V. The Society further instructed the Senior Secretary to have the Report sent to the members of the profession resident in Midlothian.

I. (*a*) That certain of the common infectious diseases, viz. CHICKENPOX, RUBELLA, and MUMPS, being of such slight importance from the points of view of mortality or serious complications, may with advantage be ignored as far as quarantine is concerned, the actual patients themselves being of course at once isolated.

(*b*) This is not meant to apply to "contacts" attending as day-scholars, or boarders proposing to return to a school which is free from such infection.

(*c*) As for all practical purposes the above-named diseases do not occur twice in the same patient, where, in the case of patients over 7 years of age, there is evidence that the "contact" has suffered from a previous attack, the above rider (*b*) does not hold.

(*d*) As the use of the term "*German Measles*" leads to serious confusion in carrying out regulations for both "Measles" and "German Measles," it is strongly recommended that the name "*Rubella*" be substituted for that of "German Measles."

(*e*) In the case of a patient contracting any of the above diseases, the isolation periods are as follows:—

(α) *Chickenpox:* when every scab has fallen off.

(β) *Rubella:* in 5 days after the appearance of the rash.

(γ) *Mumps:* in not less than a fortnight from the date of onset, and in not less than 1 week after the subsidence of all swelling in the neck or elsewhere.

(*f*) In the case of "contacts" who have not had previous attacks, the following are the isolation periods:—

(α) *Chickenpox:* in 3 weeks from the last exposure.

(β) *Rubella:* „ „ „

(γ) *Mumps:* „ 25 days „ „

II. MEASLES AND WHOOPING-COUGH

The spread of these two diseases is largely due to the fact that they are most infectious before they can be diagnosed; still, in view of their serious character, isolation of actual cases and of "suspects" should of course be promptly carried out.

A. *Measles.*

Any patient contracting the disease is not to return to school for at least 3 weeks from the date of the appearance of the rash.

In the case of "contacts" who have not had the disease, the period of quarantine should not be less than 14 days.

"Contacts" who have had a previous attack may return to school if the *patient* has been isolated, and efficient disinfection of the *contact* has been carried out. (This recommendation is subject to modification in the smaller class of houses.)

B. *Whooping-Cough.*

It appears most probable that the infectivity of whooping-cough *is associated with the catarrhal stage, and, in the absence of complications,* disappears very shortly after the development of the "whoop." It is desirable, however, for the patient's own sake that he be under careful treatment or observation till the disappearance of the "whoop."

In the case of "contacts" who have not had the disease previously, the period of quarantine should be 21 days.

"Contacts" who have had a previous attack may return to school at once.

III. Diphtheria

A patient contracting the disease must be isolated until the condition of the throat, nose, ears, etc., is normal, and until at least two consecutive negative swabs, taken at intervals of not less than 24 hours, have been obtained from the affected parts.

"Contacts" should be kept under supervision until two negative swabs have been obtained at intervals of not less than 24 hours.

IV. Typhoid Fever

A patient contracting the disease may return to school in not less than 6 weeks from the onset, and after complete recovery.

No quarantine of "contacts" is required except in special circumstances.

V. Scarlet Fever

Patients contracting the disease must be isolated for not less than 6 weeks from the date of the eruption, and until recovery from all complications *which may have a distinct bearing on the question of infectivity.*

"Contacts" must observe a quarantine of not less than 10 days after efficient disinfection.

(It being generally agreed that the incubation period of scarlet fever does not in any case exceed 5 days, the Committee (with one exception, viz. Dr Maxwell Williamson) were of opinion that contacts might safely return to school in 7 days.)

3. REPORT OF THE COMMITTEE APPOINTED BY THE MEDICO-CHIRURGICAL SOCIETY UPON THE EDINBURGH MILK SUPPLY

The terms of the remit to this Committee which was appointed by the Medico-Chirurgical Society on February 5th, 1913, were as follows: To inquire whether the milk supply of Edinburgh bears any relation to the prevalence of tuberculosis in the city, and to report to the Society.

At the date of its appointment the Committee was cognisant that certain investigations bearing upon the subject of the remit were in progress in Edinburgh, and it therefore resolved to await the result of these before proceeding to frame a report. Although the inquiries referred to are not yet complete, the results already published provide definite data towards the formation of a judgment on the question at issue.

Present Scientific Position

The question of whether tuberculosis in man is traceable to the disease as it occurs in cattle, and the question as to the part played by tuberculous milk in conveying infection have within recent years received much attention. The situation at present from the scientific standpoint may be deemed as follows.

First, the existence of two main strains of the tubercle bacillus—the human and the bovine—may be taken to have been established. While it is probable that intermediate types differing in growth characteristics and pathogenicity occur, these constitute a small proportion of the strains hitherto isolated from tuberculous infections.

Second, as the result of varied investigations in different parts

of the world, it is certain that bacilli of the bovine strain have been isolated from many cases of tuberculosis occurring in man.

Third, the relative parts played by the human and bovine bacilli in human tuberculosis have still to be determined. It is probable that pulmonary tuberculosis (phthisis) is usually due to the human strain, but very few extended series of observations on other forms of tuberculosis have been carried out. In such inquiries as have been made, too little attention has been paid (*a*) to the relative prevalence of the two types at different age periods, and (*b*) to the fact that different results may be obtained according as the tuberculous material used for investigation has been removed during life or at post-mortem examination.

The last consideration is of great importance in that, while bovine infection may have less tendency to culminate in a fatal result than when the human strain is the causal factor, it is none the less certain that the bovine bacillus is responsible for many cases of tuberculosis occurring in bones, joints, or glands. It is thus clearly responsible not only for much suffering but for the permanent disablement of many members of the community.

Up till recently the most extended series of observations on the prevailing strains of tubercle bacilli have related to tuberculosis in adults, and especially to pulmonary tuberculosis, and when the disease in children has been investigated the tuberculous material has usually been derived from the post-mortem room. At present Edinburgh is the only place where a systematic study of tuberculosis in children has been carried out on material removed by surgical operation. Here Fraser, (*Journ. Exper. Med.*, 1912, vol. xvi. p. 432), studying 70 cases of tuberculosis of bones and joints in children under twelve years of age, showed that in 60 per cent. the bovine strain was present. Further, it appeared that the incidence of the disease corresponded with the period when the children began to be fed on unsterilised cow's milk. Again, Mitchell,[1] (*Brit. Med. Journ.*, 1914, vol. i. p. 125), working under similar conditions on 72 cases of tuberculosis of cervical glands, found that in 90 per cent. the bovine strain was present; here also the incidence of the disease appeared to correspond with the commence-

[1] In the observations of both Fraser and Mitchell a proportion of the cases were children sent from the country to Edinburgh for treatment. There were, however, no essential differences in the relative prevalence of the bovine and human strains in these cases as compared with children from the city of Edinburgh.

ment of feeding with cow's milk. Mitchell in a number of cases further found that tuberculous cows were contributing to the milk supply of certain of the households from which the children came.

The Edinburgh Milk Supply

Turning to the question of direct evidence regarding the prevalence of tuberculous milk in Edinburgh, we find that adequate data of an official character on which a judgment can be formed are wanting. In answer to a question put in the House of Commons on the subject on 10th March 1914, the Secretary for Scotland quoted a statement by the Medical Officer of Health for Edinburgh to the effect that "investigations carried on for several years show that only in exceptional cases is the presence of tubercle detected in samples of milk taken in the city." So far as the Committee are aware, the only information as to these investigations which is available is contained in the Annual Reports of the Medical Officer of Health. In those for 1908 and 1909 no reference is made to the subject. In 1910 it is stated that "several" samples of country milk were taken and that the results of the examination were entirely favourable in every case. It is not stated, however, by what methods the examination was conducted. In 1911 it is stated that 22 samples from dairy farms ("a number fewer than usual") were taken, that these were tested by the inoculation method, and that no evidence of the presence of the tubercle bacillus was found. The 1912 Report contains no reference to the subject. The only other bacteriological investigations recorded have reference to cows found by the veterinary inspector to present appearances suspicious of tuberculosis. The following independent observations form a suggestive contribution to the subject. Miller, (*Edin. Med. Journ.*, 1914, vol. xii. p. 156), gives the result of an investigation of 101 samples purchased from milk shops distributed in different parts of the city, and of these 16 were found to contain living tubercle bacilli. Similarly, Mitchell, (*Edin. Med. Journ.*, 1914, vol. xii. p. 315), investigating 201 samples from certain districts of the city, found tubercle bacilli in 41 of these (*i.e.*, 20 per cent.).

There is likewise indirect evidence as to the prevalence of tuberculous milk in Edinburgh. Notwithstanding improvements in the sanitation of byres within the city, and the decrease of the number of cows kept therein, there has been during recent years little or no diminution in the number of tuberculous cows con-

demned in these dairies. It is further to be noted that during the past nine months, when a Tuberculosis Order has been partially in force in Scotland, a large number of cows have been condemned for tuberculosis in the counties from which the Edinburgh milk supply mainly comes.

Although direct evidence of the existence of tuberculosis traceable to feeding with tuberculous milk has only recently been available, there has for some years been a grave suspicion in the minds of many members of the medical profession in Edinburgh that such a source of infection prevails in the city. In the end of 1910 the views of the Staff of the Royal Hospital for Sick Children were put before a representative gathering of persons interested in the welfare of child life, and the importance of attempting to procure a more reliable milk supply in Edinburgh was recognised. Representations were made on the subject to the Public Health Committee, but apparently no action has followed.

Taking these facts into consideration the Committee is of opinion that a serious situation exists which calls urgently for action.

They are of opinion that steps should be taken to secure a more thorough and systematic bacteriological examination of the Edinburgh milk supply, so as to determine the exact extent to which it is infected with tubercle bacilli. In this connection it would be well to follow such a procedure as that adopted in Manchester, where for nearly twenty years an efficient organisation has been at work. Large numbers of samples of milk ought to be taken both from retail shops and from supplies arriving at the railway stations, and these should be examined by applying the inoculation test which is the only reliable means of examining a *mixed* milk.

Further, whenever in the course of the inspection of dairies any suspicion of the existence of tuberculosis in a cow is aroused, its milk should be examined ; in this case (*i.e.*, in dealing with the milk of a single cow), Delépine, (Supplement to 38th Report of Local Government Board, 1908-9, p. 341), has shown that by special microscopic methods a reliable opinion as to the presence of the tubercle bacillus can be arrived at in a few hours. When suspicions are confirmed the Medical Officer of Health ought to exercise his power under the Local Acts and immediately forbid the milk being sent into the city. In connection with that part of the milk supply drawn from the country, the attention of the Committee has

been drawn to the insanitary conditions under which this is often handled on arrival at the city, and as a subsidiary point the Committee would urge that steps should be taken to remove this defect.

Such measures of inspection, while they furnish the authorities with information as to the prevalence of tuberculous milk, constitute at the same time an efficient means of improving the milk supply. Not only are infected animals eliminated from herds supplying milk, but cow-keepers become chary of sending milk of which they have any suspicion to a district where they know its characters may be subjected to strict scrutiny.

The other line of action ought to be directed to increasing the means for the inspection of animals supplying milk to the city. The Committee readily recognises that there are dairies supplying milk to Edinburgh which are conducted along well-ordered lines. At the same time in many cases an opposite state of affairs exists, and to deal with these a machinery of strict supervision must be provided. The Committee is of opinion that in this direction the present administrative arrangements in Edinburgh are inadequate. Only one Veterinary Inspector, who is also a Food Inspector, is available for the work. While he is able to devote considerable attention to the 70 byres within the city boundaries, as is shown by the fact that 1126 visits were paid to these during 1912, he obviously exercises no effective control over country byres; yet it is from these that about two-thirds of the total milk supply of the city are derived. During 1912 only 20 visits were paid to these byres, and it may therefore be said that the powers vested in the Magistrates and Council for dealing with tuberculosis in country byres have been inoperative. An increase in the veterinary staff of the city and an extension of the work of the veterinary inspectors is thus urgently demanded.

The efficiency of such lines of action as those just suggested for improving the milk supply is shown by the experience of Manchester where, while in 1807-08 17 per cent. of the milk was tuberculous, it was found that (though the figures fluctuate from year to year) the average for the years 1900-07 was only 0 per cent. and for the years 1907-09 0.36 per cent. A marked improvement has followed from the systematic reinspection of individual herds.

There is still a wider aspect of the matter. The recent Tuberculosis Order of the Board of Agriculture gives a measure of power

of compulsory slaughter, but in the case where an Edinburgh veterinary inspector has detected a tuberculous cow in a country herd its destruction could only be effected through the Local Authority which has jurisdiction in the place where the animal was found. It is obvious, therefore, that in addition to the measures already suggested it is necessary to secure co-ordinate action between different local authorities. This would to a large extent be achieved by the passing of such a Milk Bill as that which has already been several times before Parliament.

Conclusions

The general conclusion of the Committee after consideration of all the facts is that infection of children in Edinburgh with the bovine bacillus derived from milk is prevalent. From the absence of data it is impossible to say whether the disease is more common here than in other cities under similar conditions.

The Committee is further of opinion that the means taken by the Edinburgh Public Health authorities to secure a non-tuberculous milk supply for Edinburgh are at present insufficient.

The Committee believes that at present the only means by which the consumption of milk in Edinburgh can be rendered certainly safe, is through its being sterilised by boiling at the hands of the consumer.

They are aware that two main objections have been raised to that procedure, namely, firstly, that thereby the nutritive qualities of the milk are seriously impaired, and secondly, that the sterilisation process destroys certain qualities of the milk and renders children nourished upon it liable to a form of scurvy.

With regard to the first of these, the Committee is of opinion that the objection is groundless. It has arrived at this conclusion from a consideration of the evidence collated by Lane-Claypon. (Reports to the Local Government Board on Public Health and Medical Subjects, New Series, No. 63, H.M. Stationery Office, 1912), and of similar facts which have come under the observation of its own members and of others whom it has consulted.

With regard to the second objection the Committee apprehends the position to be as follows. If young children during the period when they ought to be nursed by their mothers are fed exclusively

on boiled milk, in a certain small number of cases a degree of scurvy may result, but the condition is a slight one and readily yields to simple treatment. In children from the time when they begin to be nourished on a properly adjusted mixed diet, infantile scurvy is practically unknown.

It has been stated that children fed on boiled milk are more liable to rickets than where raw milk is used, but in the opinion of the Committee no evidence exists pointing to such an occurrence.

Within recent years the argument has been advanced that it is actually a benefit to a child to be fed on the milk of tuberculous cows when this is mixed with a large quantity of tubercle-free milk, in that the children are thereby immunised against tuberculosis and are less liable to infection with the virulent human strain of the bacillus in later life. In the opinion of the Committee this contention is at present unsupported by scientific evidence, and the arguments advanced have been largely of an academic nature. In any case the Committee is of opinion, firstly, that the risk of serious disease being contracted from tuberculous milk must be put against the supposed immunisation effected; and, secondly, that if it were established that the bovine bacillus can immunise an individual against subsequent infection with the human bacillus, there are safer means by which this immunisation can be accomplished than by using living tubercle bacilli given in milk.

Recommendations

The Committee recommend as follows —

Firstly, that the members of the Medico-Chirurgical Society use every opportunity meantime to urge upon heads of households the advisability of boiling all milk consumed in their families.

Secondly, that a strong representation be made to the Town Council with a view to the reorganisation of the administration regarding milk examination and the inspection of cows within and without the city.

Thirdly, that the Government be urged to carry through at an early date a Milk Bill along the lines of the Bills which have been under the consideration of Parliament.

The Framing of the Report

In addition to those whose signatures are attached to the Report, the Committee included Sir R. W. Philip, who took part in the deliberations and in framing the Report. In view of his recent appointment as Expert Adviser to the Corporation regarding Tuberculosis, it seemed proper that he should not be a signatory to the Report.

James Ritchie,
Convener.
Harold J. Stiles.
A. Dingwall Fordyce.
James Miller.
John Fraser.
A. Philp Mitchell.

June 1, 1914.

The above Report was considered and finally adopted by the Medico-Chirurgical Society of Edinburgh at a meeting of the Society held on July 1st, 1914.

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